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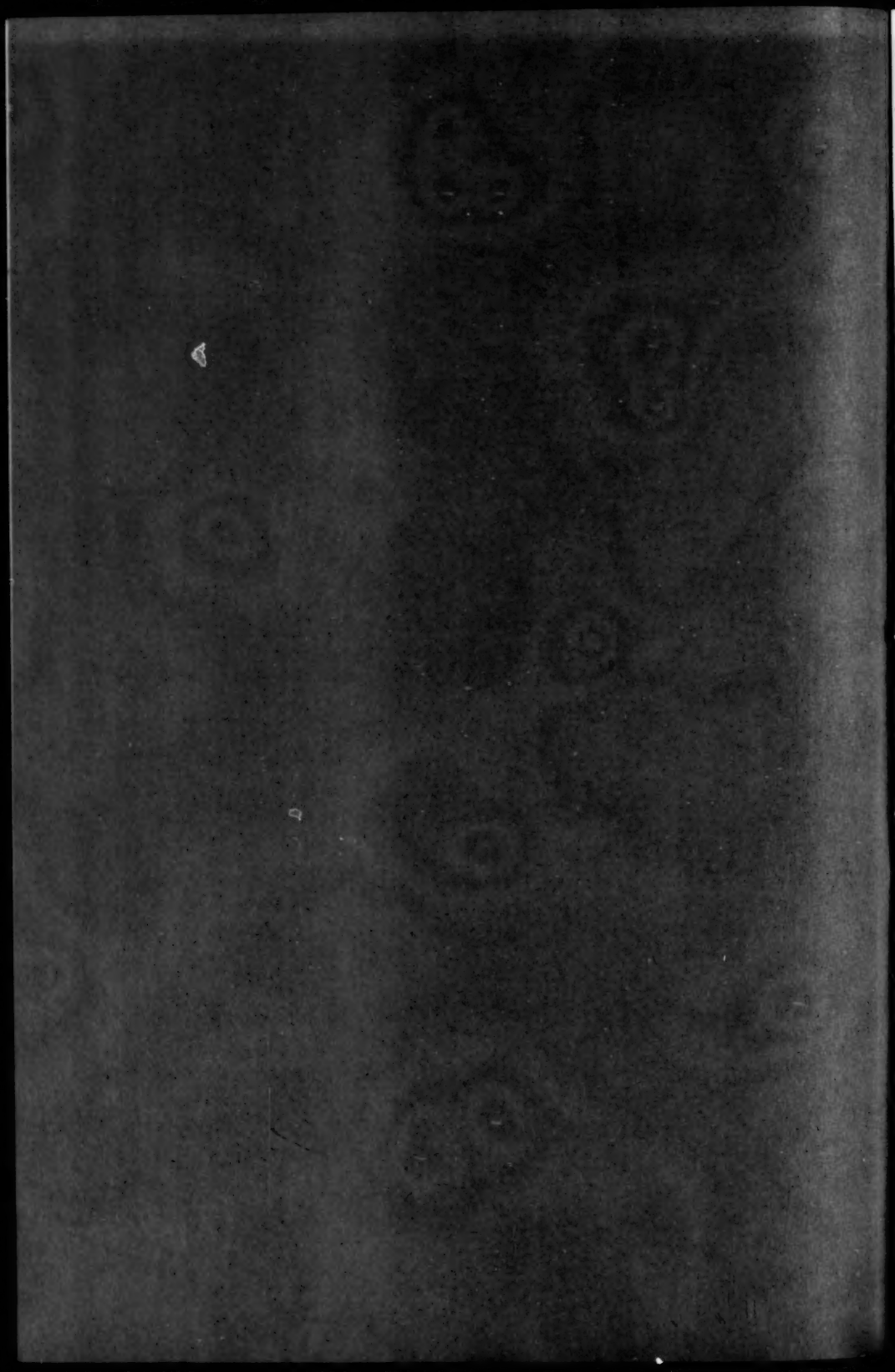

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ARTERIOSCLEROSIS*

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WE have increased the expectancy of life from forty years at birth in 1850 to sixty-three years at the present time. This increase in life expectancy is due largely to the control of infectious diseases of childhood and early adult life. We are an ageing population, among whom chronic non-infectious diseases, notably diseases of the cardiovascular system, are becoming more important. The medical handling of these diseases includes little hope of cure, since a disease is usually well-established at the time of its discovery. The principal promise lies in the direction of prophylaxis. Prevention can be applied most intelligently when we have specific evidence of the cause of a given disease. Concrete evidence in this respect is now available with reference to the most important chronic disease, atherosclerosis.

The term arteriosclerosis is properly limited to non-infectious chronic disease of the arterial system. Arteritis, whether syphilitic, rheumatic, typhoidal or related to other infections, thrombo-angiitis obliterans, periarteritis or polyarteritis nodosa and productive thrombotic diseases are excluded.

* From the Medical Examiner Service, Suffolk County, Mallory Institute of Pathology, Boston. This work was aided by grants from the Committee on Scientific Research, American Medical Association. Read October 14, 1941 in the Graduate Fortnight of The New York Academy of Medicine.

We are left with the arteriolar (diffuse) sclerosis, Mönckeberg's sclerosis, and atherosclerosis. Arteriolar sclerosis is characterized by (1) hyaline intimal lesions which are analogous to those occurring in atherosclerosis, by (2) hypertrophy and degeneration of the media, by (3) proliferative thickening of the subendothelial layer of the intima, and is closely allied with hypertension.[1] There is also a close relation between hypertension and atherosclerosis, particularly with reference to the coronary arteries.

Mönckeberg's sclerosis, responsible for beaded and pipe-stem arteries, is not clinically significant. The media is calcified in the relaxed state resulting in dilatation of the vessels. Clinical disturbances are due to engrafted atherosclerosis.

Atherosclerosis is responsible for most of the arteriosclerosis which is clinically significant. It was named mushy sclerosis literally by Marchand because of the fatty—mushy—material in the lesions. Adami and Aschoff[2] demonstrated that the fatty material was made up of myelin bodies, later shown to be cholesterol esters. Anitschkow and Chalatow[3] produced the disease in rabbits by feeding cholesterol. The experimental evidence was not generally accepted and the experimental disease was referred to, disparagingly, as the cholesterol disease of rabbits. All active human atherosclerotic lesions contain excess cholesterol. Only healed (scarred) or calcified lesions are free from this excess. In other words, active atherosclerosis cannot occur without cholesterol.

Cholesterol is a hydro-aromatic secondary alcohol which can unite with fatty acids to form esters. It is contained in all animal cells in normal amounts, hidden in the cells, and disclosed only on chemical examination. All visible cholesterol is excess cholesterol. Excess cholesterol is present in atherosclerotic lesions in two forms: esters which occur in droplets—the so-called fluid crystals of Lehmann—and solid notched oblong plates. Both fluid and solid crystalline forms are anisotropic (doubly refracting) and permit of identification of the substance in the lesions.

Phagocytic cells pick up excess cholesterol esters, much as they would engulf particulate solid matter. These cells, loaded with fine ester droplets, are known as foam cells. Though other lipoids—kerasin, sphingomyelin—may be found filling foam cells in the rare lipoidoses, 90 per cent at least of foam cells in human tissues carry cholesterol esters.

Cholesterol was looked upon by Starling as the stable substance of

the animal cell, in whose interstices the more labile substances underwent metabolism. With ergosterol it is the only sterol capable of absorption through the intestine. Vegetable sterols are not absorbed. The dietary sources of cholesterol are egg yolk, milk and meat fats. Cholesterol is the source of the sex hormones and adrenal corticosterone; is closely related to the bile acids, the fat soluble vitamins and the carcinogens. Its metabolism appears to be under thyroid control. Females mobilize cholesterol during pregnancy, and appear to possess a better cholesterol metabolism than do males.

Cholesterol is an irritant. It is, however, relatively benign, provoking chronic irritant effects. In this respect it is akin to silica. Gye and Purdy[4] injected silica sol (colloidal silica) intravenously into rabbits, and produced after long delay a characteristic triad of lesions, cirrhosis of the liver, enlargement of the spleen and changes in the kidney characteristic of chronic "interstitial" nephritis. I have been able to demonstrate that adequate amounts of cholesterol fed to rabbits will produce after long delay, in addition to atherosclerosis, cirrhosis of the liver, enlargement of the spleen and chronic vascular nephritis.[5] Both silica and excess cholesterol are difficult of metabolism, tend to persist in the tissues for long periods, and stimulate a growth of connective tissue.

The Lesions of Atherosclerosis

According to the Virchow-Aschoff theory, the wear and tear on the vessels by pressure of the circulating blood produces a swelling of the deep layers of the intima accompanied by degeneration. The arterial intima is not vascularized but depends for its nutrition on imbibition or diffusion of plasma through its tissues from the blood circulating through the lumen. Cholesterol esters, according to this theory, are precipitated from the plasma in its passage through the swollen intima and occur as free esters in the lesions. These free esters are picked up by phagocytic cells from the intima, which constitute the foam cells found in the lesions.

Klotz[6] insisted that the earliest lesions of atherosclerosis were marked by the presence of foam cells. There was no swelling or degeneration of the intima, and no free cholesterol esters. He believed that the description of Aschoff applied to advanced lesions. The free esters were due to the disintegration of foam cells as a part of the progress of the lesion.

My studies of early lesions have supported Klotz' findings. The earli-

est lesions show foam cells in the subendothelial layer of the intima. There is no swelling or degeneration of the intima, and there are no free esters.

Atheroma. In the early youth the stay of foam cells in the subendothelial layer of the arterial intima may be temporary. A mechanism which I have described[7] removes the excess cholesterol from the lesions and the tissues are restored to the normal. This power to remove excess cholesterol rapidly from the tissues is gradually lost as age comes on but persists in the ascending aorta into old age.

Atherosclerosis. Progressive lesions, i.e., those of true atherosclerosis, are marked by stimulation of connective tissue growth due to the excess cholesterol in the foam cells in the lesion; invasion by the foam cells of the deeper layers of the intima; necrosis of the deep layers as the lesions enlarge beyond the power of plasma diffusion to support them; vascularization; repair with scarring; or calcification. In very susceptible persons in youth, and as a standard lesion in older ages, foam cells may pile up in masses without adequate provision for nutrition or for physical support of the cells. Massive necrosis of these cells gives rise to lesions with soft mushy contents whose thin covering layer tends to rupture at a touch. These lesions are improperly called atheromatous abscesses. They are actually antitheses of abscesses. There is no cellular exudate and the concentrated cholesterol prevents infection. A better term for such a lesion is atherocheuma (literally mush liquefied). Atherocheumas may rupture producing atheromatous ulcers, which may give rise to thrombi and, rarely, to dissecting aneurysms.

Nodular lesions of the types mentioned above are characteristic of human atherosclerosis. The progressive steps in the evolution of the varied gross lesions of the human aorta can be followed with considerable accuracy.[7] The response to fat stains and the anisotropic property of the excess cholesterol together with the distinctive appearance and staining reactions of the foam cells make such a study possible.

Coronary Sclerosis

The frequency with which lesions of the coronary arteries occur and the serious character of the lesions are in all probability due to the unusual stresses which these vessels are called upon to bear. The coronary orifices are located just above the aortic ring in a region exposed momentarily to maximum stress by the closure of the aortic cusps. The pressure

is quickly relieved by diffusion through the elastic aorta but must have an influence on the structures subjected to it. Furthermore, the coronary branches are distributed through the muscle bundles of the heart. In experimental animals the ventricular wall can be seen to become pale during systole, due to the compression of the muscular arterial branches, the capillaries and the veins. Meantime the primary coronary vessels and the main subepicardial branches, not subjected to compression, are filled with blood under systolic pressure and cannot be emptied. The principal coronary circulation is during diastole.

Probably because of these unusual stresses, the coronary arteries develop, beginning in the "teens" or earlier, a thickened intima in the form of what I have called a "buffer" layer. Other vessels, notably the proximal portions of the intercostal arteries, the hepatic artery, and the vessels of the penis, uterus and ovaries, subjected to stresses, develop thickened intimas, unrelated to arterial disease as such. But in none of these vessels is the thickening so constant and so perfect a part of the vessel as is true in the coronary arteries.

The localization of the lesions of coronary sclerosis is due to stresses and is most constant in the proximal 3 to 4 cm. of the left coronary artery and its descending branch. Not only are the lesions most constant in this site but they tend in general to progress toward occlusion more rapidly here. The left coronary artery in this region turns almost at a right angle from its origin and gives off almost at a right angle a very large branch, the circumflex. The right coronary artery does not undergo such a change in course and does not give off a large branch so early. However, as atherosclerosis progresses both coronary vessels and many of their branches are affected. In advanced cases the distribution of the lesions may be essentially equal in both coronaries.

Coronary sclerosis furnishes the best evidence that atherosclerosis is not a disease of age. In my series advanced coronary sclerosis at 12 and 15 years of age and coronary deaths at 24, 25, 26, 28, 29 years of age, with many in the thirties, determine that issue beyond question.[8] Many of the young victims were robust athletic types. There were no stigmata of senility in any of the younger group.

Coronary lesions tend to vary in their histologic makeup with the age of the individual. The initial invasion of the subendothelial layer of the intima by foam cells is followed by stimulation of connective tissue and thickening of the intima. The foam cells invade the deep layers of the

thickened intima. New waves of foam cells invade the lesion as it enlarges. Necrosis of the deep layers, which are furthest removed from the lumen, is usual. If the lesion is to enlarge, a new source of nutrition must be found. Capillaries arising in most cases directly from the lumen supply this need. In some cases extensions of the vasa vasorum furnish the new circulation. In *youth* the capillary system lies in a relatively loose textured connective tissue. Rupture of the delicate capillaries may result from compression by coronary spasm. (Hall and his associates[9] have produced coronary occlusion with infarct production, and also coronary thrombosis—without atherosclerosis—by the use of acetylcholine, which provokes coronary spasm.) Hemorrhage from capillary rupture is followed by fibrin formation and fibrinoid necrosis. This may extend to the lumen and thrombosis may follow. Thrombosis is the usual termination of coronary sclerosis in youth.

In the *middle period* of life there is a greater tendency to collagen production, and scarring of the lesions is outstanding. The new accretions of foam cells are perhaps less frequent than in the group who die early in life. Narrowing of the lumen by the encroaching lesion is on the whole slower in this period. Hypertension is a common associated condition. As the lumen narrows a state of coronary insufficiency may result. In a large percentage of members of this group death of coronary type may occur without occlusion of either main coronary artery or of any branch of a size adequate to account for so rapid a death. Indeed the individual may undergo an acute coronary seizure; may be hospitalized for days before he dies; and postmortem examination may disclose an infarct with no occlusion of main vessels or of branches supplying the infarcted region. Spasm alone can account for such a lesion. Rarely one finds in this group a well vascularized coronary lesion with a delicate capillary system, with capillary rupture and thrombosis—the lesion associated normally with younger individuals. Calcification of the arteries following necrosis in atherosclerotic lesions is common but scarring is the main characteristic of the lesion in this period.

In *older ages* the common lesion is one of atherocheuma formation. The reaction of connective tissue to the presence of cholesterol is less marked. Enough connective tissue to support the massed lipoid cells may not have been produced. Death may result from coronary insufficiency, but frequently death is due to the rupture of an atherocheuma. The pasty contents of an aortic atherocheuma are broken up in the whirling

blood stream and can produce at most capillary embolism. The pasty contents of a coronary atherocheuma can cause immediate occlusion of the lumen. Secondary thrombosis can occur when death is delayed. Scars from older lesions are common in old age, as is calcification.

Continuity of heart action is, of course, essential to life. As Lewis has demonstrated, muscular action without adequate oxygen is responsible for pain, and accounts for angina. The oxygen requirements of heart muscle are greater than those of other tissues excepting the brain. As coronary disease narrows the vascular lumen the common cardiac lesion is chronic vascular myocarditis or myocardosis. As the blood supply is cut down the oxygen-demanding muscle fibers undergo atrophy and, if the interference with supply progresses, the fibers disappear. The tougher connective tissue which replaces them proceeds rapidly to scar formation.

At the other end of the scale we have the true infarct due to a sudden cutting off of the blood supply by thrombosis, rupture of an atherocheuma or spasm. In this case we have necrosis not only of the muscle fibers but also of the supporting connective tissue, followed by acute inflammatory exudation.

Midway between these lesions and apparently due to spasm is a process of rapid necrosis of muscle fibers in small regions, but followed by a stimulation of the supporting connective tissue, associated with little or no inflammatory reaction. These lesions have been called miliary infarcts. They are not true infarcts. A better term is miliary myocardoses.

Rupture through the necrotic tissues of an infarct may cause death by hemopericardium and cardiac tamponade. Rupture of an atherosclerotic coronary artery may lead to hemopericardium. Dissecting aneurysms of the coronary arteries are rare. Infarcts are repaired by granulation, sometimes with pericardial adhesions. The scar tissue repairing an infarct may stretch producing a cardiac aneurysm. Thrombi undergo organization, if the individual lives, sometimes with canalization. Rarely the canalization may reëstablish the circulation in considerable part. Usually the blood flow is reëstablished about the region of thrombosis by a collateral circulation.

Coronary occlusion (1) may be symptomless; (2) may cause mild or serious illness; or (3) sudden death with ventricular fibrillation; or (4) death so instant as to suggest immediate cerebral arrest with cardiac and respiratory standstill.

Experimental Atherosclerosis

Just as the primary stage of human atherosclerosis is marked by the presence of foam cells in the subendothelial layer of the intima, so in the primary stage of experimental atherosclerosis in the rabbit foam cells appear in the subendothelial layer of the intima. This suggested a search for the source of these foam cells.[5]

There is a period of weeks following the beginning of cholesterol feeding in the rabbit before the first lesions appear in the ascending aorta. The reason for this delay—this latent period—was made clear by feeding cholesterol to a large series of animals and killing an animal on every second day in the critical period beginning with the fifth week. In frozen sections of the liver stained with Sudan IV the blood, rich in cholesterol after several weeks' feeding, stained deeply. The fat was limited to the blood. In animals killed later isotropic fat could be seen in the cells in the centers of the liver lobules. Later anisotropic fat appeared in the form of cholesterol esters in central cells, and began to accumulate in the liver cells (see footnote).* In animals allowed to live for longer periods the cholesterol esters accumulated in liver cells until a large part of the cells of the lobule were filled. In these animals the adrenal glands also began to show visible enlargement. As the amount of cholesterol esters in the liver and adrenal cells became greater there came a point at which Kupffer cells in the liver and reticulo-endothelial cells from the capillaries of the adrenals began to remove the excess esters from these organs. The esters were picked up by these cells as though they were particulate matter.

In animals which had received a great excess of cholesterol and had lived well beyond the latent period, the overloading of the liver and adrenal cells with esters reached such a degree that the liver sinusoids and the adrenal capillaries, as also the lymphatics, became blocked with phagocytic cells loaded with cholesterol esters. As a result the procedure of removal of the excess esters was so slowed up that the details of the process could be followed. The cells loaded with esters, now become foam cells, made their way into the lymph and blood stream. They were carried in the circulation to the lungs. They passed through the wide lung capillaries apparently with ease. The so-called filter of the lungs is

* It has been assumed that the liver esterified cholesterol since Thannhauser and Schaber[10] in 1926 demonstrated that in "parenchymatous" liver disease there was a lowering of the ratio of ester to free cholesterol in the blood. We now have optical evidence of liver esterification of cholesterol. Esterification of cholesterol was observed by Chalatow[11] in the cells of the adrenal glands in 1922.

efficient for larger masses such as megakaryocytes; but foam cells, ameboid and fitted to make their way through the interstices of solid tissue, found no difficulty in the passage. Having passed the lungs the cells invaded the subendothelial layer of the intima of the aorta. This invasion of the arterial intima must be due to a positive chemotaxis, since cells carrying particulate matter do not tend to invade the aorta or arterial intima. The only exception to this rule is the invasion of the aortic intima by cells carrying polyvinyl alcohol.[12] The chemotaxis and arterial invasion by foam cells may thus be a type reaction, limited to the higher alcohols.

The foam cells which mark the first stage of atherosclerosis in human and experimental lesions have now been brought from origin to destination in the experimental rabbit. It is a reasonable assumption that the mechanism of atherosclerosis production in man is the same as that seen in the experimental animal, though in less dramatic form. The progress of human atherosclerosis is so slow that the formation of foam cells in the liver must be only occasional and perhaps localized within this relatively enormous organ.

The lesions of atherosclerosis in the experimental rabbit duplicate much more exactly human lesions than do the attempts to reproduce in animals the lesions of many human infections by the use of their known causal agents. Such differences as are found between human and rabbit atherosclerotic lesions are dependent largely upon differences in the method of cholesterol feeding—in a single continuous sequence over a period of months in the rabbit—intermittently at intervals over a period of years in man.

Since cholesterol is the cause of atherosclerosis the disease is preventable. There are indications that the human body can synthesize the cholesterol it needs. The substance is apparently required in greatest amount during periods of active cell production. The cholesterol of egg yolk is needed for the embryo. The cholesterol of milk is required by the nursling after its intrauterine supply from the blood of the mother has stopped. At any rate the needs of the body for this substance must be less after the period of rapid cell growth has passed. Prevention by diet should be more successful than is true of diabetes, since cholesterol does not appear to be a necessary part of the diet. Vegetable fats, whose sterols are not absorbed, may be substituted for animal fats.

Choline and lipocaic, which are said to prevent the excessive deposit

of fat in the livers of experimental animals, may influence the deposit of excess cholesterol in the arteries. The evidence in that direction up to now is not very strong, however.

Summary

Atherosclerosis, the important form of arteriosclerosis, is distinguished from other forms of arteriosclerosis by the presence of excess cholesterol in the lesions. It is the "cholesterol disease" of man.

Excess cholesterol, i.e., visible cholesterol, is an irritant, producing lesions in the experimental rabbit (in addition to atherosclerosis) resembling those produced by intravenous silica.

Earliest lesions of atherosclerosis in the experimental rabbit and man are marked by the presence of foam cells containing cholesterol esters in the subendothelial layer of the normal arterial intima. This lesion regresses in atheroma by a mechanism of cholesterol removal. It progresses in atherosclerosis to produce the characteristic nodular lesions of the disease.

In the experimental rabbit, fed cholesterol, it has been possible to observe the esterification of cholesterol in the liver and the adrenals; deposition of these esters in liver and adrenal cells to the point of becoming a burden; engulfing of the excess esters and their removal from the liver and adrenals by Kupffer and corresponding cells; escape of these cells into the circulation; their passage of the lungs and invasion of the arterial intima. Thus are begun new atherosclerotic lesions, or accretions are made to those already started.

Excess cholesterol is the cause of atherosclerosis. Stresses determine the localization of lesions. Thyroid secretion controls cholesterol metabolism. Sex, age (time + thyroid deterioration) and heredity are modifying factors.

Diet, with limited or absent cholesterol, should prevent atherosclerosis. Vegetable oils, whose sterols are not absorbed, can be substituted for animal fats.

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CORONARY INSUFFICIENCY: OBSERVATIONS ON DIAGNOSIS AND TREATMENT*

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CORONARY insufficiency is a functional disorder, not dependent upon any specific anatomic lesions for its occurrence. It may be defined as that condition in which the coronary arteries deliver less blood than is required for the effective performance of the heart. The disproportion between supply and demand can be brought about by a number of different factors which either diminish coronary flow or increase cardiac work. Its commonest cause is disease of the coronary arteries. Its two most important effects are anoxia and ischemia of the heart muscle. According to the current concept acute, local anoxia is the chief cause of cardiac pain. Prolonged ischemia, in the sense of a permanent reduction in coronary flow, may induce fibrosis of the heart muscle, cardiac hypertrophy and, eventually, congestive failure. In this discussion cardiac infarction and congestive failure will not be considered. Attention will be focussed on the paroxysmal type of pain to which the term "anginal" has been applied.

CAUSES OF CORONARY INSUFFICIENCY

Before proceeding to a consideration of diagnosis and treatment, it is necessary to have clearly in mind the various clinical conditions which may result in an inadequate coronary flow. Among the most important are:

1. Diseases of the coronary arteries.
    A. Atherosclerosis. This may be
        a. Slight, with patchy intimal change and loss of elasticity.
        b. Marked, with calcification and narrowing.

* Presented before the Graduate Fortnight of The New York Academy of Medicine, October 14, 1941. From the Department of Medicine, College of Physicians and Surgeons, Columbia University, and the Medical Clinic of the Presbyterian Hospital, New York.

c. Occlusive, as a result of obliteration of the lumen by the sclerotic process or due to the formation of a thrombus.

B. Syphilis of the aorta, with stenosis of one coronary orifice, or of both. The specific process is limited to the first 3 or 4 mm. of the coronary arteries.

C. Embolism. This occurs infrequently; the two most common sources of emboli are bacterial vegetations on the mitral or aortic valve, or a thrombus covering a sclerotic plaque at the root of the aorta.

D. Rheumatic fever, with involvement of the coronary arterial walls and, occasionally, thrombus formation.

E. Periarteritis nodosa, a relatively rare condition, but one in which coronary involvement is not uncommon.

2. Aortic valvular disease. In aortic stenosis, less frequently in aortic insufficiency, the coronary blood flow may be diminished. In free aortic regurgitation this is believed to be due to the lowered diastolic pressure.
3. Anemia. The heart suffers from anoxemia along with the rest of the body.
4. Paroxysmal tachycardia. The rapid rate may call for a greater coronary flow than can be delivered.
5. Hyperthyroidism. The increased metabolic demands of the body require a faster velocity of blood flow and an augmentation of volume output by the heart. Cardiac work may exceed coronary reserve.
6. Combined states. For example, a person with coronary sclerosis will experience pain more readily if marked anemia is present than if the oxygen-carrying capacity of the blood is normal. Ischemia and anemia complement one another in causing anoxemia.

It is thus evident that pain of coronary origin may result from systemic disorders as well as from those which are primarily cardiac. But there are other conditions which, on occasion, induce pain in the chest, and simulate that due to coronary insufficiency. Among these are:

1. Acute, serofibrinous pericarditis.
2. Aneurysm of the thoracic aorta.
3. Cholelithiasis.

4. Duodenal ulcer.
5. Poisoning by coffee, tea or tobacco.
6. Intercostal neuralgia or myalgia.
7. Psychoneurotic states.

The aim of diagnosis is to determine the etiology, to define the anatomic lesions and to estimate the degree of functional impairment. No attempt will be made here to outline a complete diagnostic survey or to give a full account of therapeutic procedure. These remarks deal with scattered observations, assembled over a period of years and concerned chiefly with cardiac pain due to coronary sclerosis.

Diagnosis

In no other condition is the patient's account of his discomfort of greater importance. Often this suffices for diagnosis. So, it is essential that the physician who is responsible for the management of the case should himself take the history. A story obtained by a colleague, no matter how competent he may be, cannot convey the same impression as that obtained from a personal interview. From this contact is often obtained the key to the entire situation. There is established an understanding and a relationship which is never gained through an intermediary. Furthermore, pain is so frequently the only evidence of coronary disease that its precise description by the sufferer must be heard in order that its significance can be appraised and its implications analyzed.

The physical examination may be entirely negative. The heart is often not enlarged, the sounds are normal and the blood pressure is not elevated. The retinal and peripheral vessels may show no sclerosis. Of positive value, though in no sense etiologically specific, are cardiac enlargement and weak heart sounds. Sometimes the first sound at the apex is split; a diastolic gallop may be heard. The presence of hypertension is not, of itself, a decisive feature.

The same viewpoint obtains with respect to the electrocardiogram. A normal record does not rule out the presence of advanced and, sometimes, serious coronary insufficiency. A case has previously been reported,[1] in which a patient with a typical history of anginal pain presented no objective signs of cardiac disease. The four-lead electrocardiogram showed no characteristic changes in T waves, RS-T segments or in conduction. Yet within twenty-four hours, this man of 49 died suddenly at the breakfast table. The presence of abnormalities in the elec-

trocardiogram is, however, of great help in furnishing positive evidence of myocardial damage.

Not infrequently, it is difficult to decide whether a person complaining of pain in the chest is suffering from coronary insufficiency or from one of the conditions simulating it, having its origin either within the thorax or below the diaphragm. To aid in differentiation and in detecting latent coronary insufficiency, the "anoxemia test" has been devised and, in our hands, has proved to be of practical value.[2, 3]

In principle, this test consists of permitting the patient to breathe a mixture of 10 per cent oxygen and 90 per cent nitrogen for 20 minutes, or until cardiac pain appears. Measurements of electrocardiograms taken at intervals during this period reveal, in patients with a diminished coronary reserve, characteristic changes which are not observed in the presence of an adequate coronary blood flow. A positive test may thus be regarded as a sign of coronary insufficiency; but a negative test does not exclude the presence of disease of the coronary arteries. The occurrence of pain during a negative test, that is, one in which no electrocardiographic changes have occurred, affords *presumptive* evidence of a diminished coronary reserve.[4] It is of particular significance when the pain appears during the first 10 minutes of induced anoxemia. Patients experiencing painful discomfort during a negative test should be carefully observed for further signs of coronary disease and managed conservatively.

The criteria of a positive reaction have been evolved after experience with the performance of the test more than 1100 times in over 500 persons, some with normal hearts, others with cardiac disease. They are as follows:

1. The arithmetical sum of the RS-T deviations in all four leads (I, II, III, and IVF) totals 3 mm. or more.

2. There is partial or complete reversal of the direction of T in Lead I, accompanied by an RS-T deviation of 1 mm. or more, in this lead.

3. There is complete reversal of the direction of T in Lead IVF, regardless of RS-T deviation.

4. There is partial reversal of the direction of T in Lead IVF, accompanied by an RS-T deviation of 1 mm. or more, in this lead. Of the four, this criterion is of the least value. It rarely occurs alone, and has been noted twice in borderline cases.[5] If further experience confirms these observations, it will be discarded.

The anoxemia test is simple and safe. Unpleasant reactions have been observed, such as vasovagal attacks, panic, loss of consciousness and dyspnea. These were relatively uncommon and not serious. In the course of 4 years, there has been no evidence of permanent injury to a single patient, although the test has been performed in the same person as often as 20 times. Early in the work, pulmonary edema occurred 3 times. But if the test is not given to a patient with congestive heart failure, to one who has had an attack of cardiac infarction within 4 months or to the same patient twice within 24 hours, no grave accidents are to be anticipated. The percentage of oxygen in the gas mixture should be checked at frequent intervals to be sure that the proper concentration is being supplied.

The test has been helpful in the differential diagnosis of conditions producing pain in the chest. It has been employed to study, in patients, the effect of various drugs on the coronary circulation. It has been useful in following variations in the coronary reserve and so appraising the efficiency of the coronary circulation at the time of its performance.[4]

The following case record serves to illustrate the type of changes which are seen in the electrocardiogram when coronary insufficiency is present, and portrays graphically the increase in coronary reserve coincident with clinical improvement.

Case I. W. D., aged 48 years, foreman in a U. S. Post Office, was admitted to the hospital on March 18, 1939. He had been well until a week before, when he had a typical attack of coronary occlusion with cardiac infarction. During this week he had been attended by his own physician. He was transported by ambulance and arrived in good condition.

On admission, the rectal temperature was 100.4° F. The heart was a little enlarged. The rhythm was regular; the rate 64. The first sound at the apex was split. The blood pressure was 120/80. The leukocyte count was 7,880, with 75 per cent polymorphonuclears. The sedimentation rate was 45 mm. in one hour, and rose on the following day to 54 mm. The Kline test of the blood was negative. An electrocardiogram showed prolonged auriculoventricular conduction (the P-R interval measured 0.33 sec.). Other changes were characteristic of a posterior infarct.

The patient remained in the hospital for 5 weeks. The course was one of steady improvement, both clinically and in the form of the electrocardiogram. At the end of another 2 weeks (8 weeks after his attack)

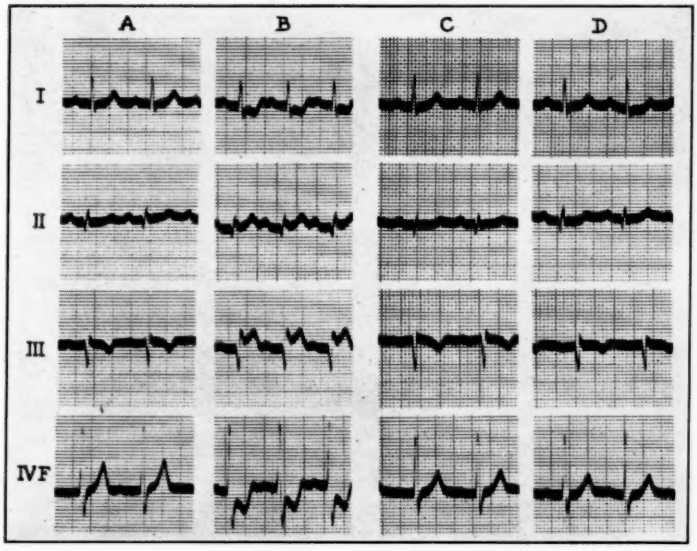

Fig. 1. *Case 1.* W. D., male, aged 48 years, foreman in Post Office. Coronary occlusion with myocardial infarction on March 11, 1939. Anoxemia test, at first positive, became negative.
Five months after attack. Working too hard and having frequent attacks of anginal pain. A—control. B—positive test: pain after 18 minutes; total RS-T deviation, 9.5 mm.; T wave reversal in Leads I and IVF.
Nine months after attack. Work lighter and nearer home. Rare attack; no nitroglycerine required. C—control. D—negative test; no pain during 20 minutes of anoxemia.

he returned to work, in spite of having been advised to extend his period of rest. When tired, he complained of aching in the left shoulder and arm. Subsequently he experienced cardiac pain on effort and in August, 1939 (5 months after the occlusion) was having frequent anginal attacks. An anoxemia test at this time was positive (Fig. 1. A, B).

He was then able to transfer to a Post Office nearer his home and was made a supervisor. The work was lighter and he improved. Nine months after the original attack, he was not taking nitroglycerine and could walk for 6 or 7 blocks before experiencing discomfort. The heart sounds were of good quality. The blood pressure was 120/72. The anoxemia test at this time was negative (Fig. 1. C, D).

The test does not furnish evidence which makes it possible to predict the future occurrence of coronary occlusion. Several patients have

suffered such attacks within a few months of the time that a negative test was found. On the other hand, patients with a positive reaction are more likely to have this complication than those in whom the reaction is negative. The test is therefore an index, within undefined limits, of the functional efficiency of the coronary circulation; but it yields no information as to the nature and extent of the pathologic lesions in the heart.

Treatment

Medical Measures. Any person who has pain in the region of the heart is alarmed. He thinks at once of a relative or friend who has died suddenly following a similar complaint and his first question is apt to be: "Have I angina pectoris?" He wants, above all, reassurance. It seems to me good practice, under these circumstances, to state at the outset that there is no disease such as angina; that this term means merely a painful or uncomfortable sensation in the chest, and that it will be our aim to discover the nature of the condition which lies at the basis of the discomfort.

These patients are worthy of the expenditure of much time on the part of the physician and attention to detail is necessary if therapy is to succeed. A carefully planned regimen, designed to save the heart from overwork, will aid in the establishment of a collateral circulation in the coronary bed. The intelligent use of nitroglycerine, not only for the relief of pain when this occurs, but prophylactically, when some necessary effort is anticipated, will prevent the occurrence of myocardial anoxia. There is evidence, both clinical and experimental,[6, 7] that repeated and prolonged impairment of the coronary blood flow causes minute focal necroses in the heart muscle and, in time, replacement fibrosis. The avoidance of painful attacks is, therefore, of therapeutic importance.

Of the various drugs which have been used in treatment, the xanthines have received most attention. There has been difference of opinion with respect to their efficacy as coronary dilators; and conflicting reports have appeared concerning their usefulness in lessening the frequency and intensity of anginal paroxysms. The changing conditions in the coronary circulation as a result of disease and the many external factors which may modify the clinical course of patients subject to attacks of cardiac pain, make difficult a just appraisal of the effect of medicinal remedies which exert their action over a long period of time.

For this reason, an objective test was applied to the problem in the form of induced anoxemia.[8, 9] Changes in the form of the electrocardiogram were studied in patients with coronary sclerosis, before and after the administration of different members of the xanthine group. The time of appearance of pain during anoxemia was also observed and variations due to drugs were noted. In brief, it was found that various members of the xanthine group (aminophylline, theophylline with sodium acetate and theobromine with sodium acetate) in proper dosage, caused a decrease in the RS-T deviation of the four-lead electrocardiogram and prolonged the time of appearance of pain. These results were obtained by averaging the observations obtained in a group of patients and expressing the effects as a trend. This was necessary because not all patients respond favorably, as might be anticipated from the varying character of the coronary lesions in different hearts. In general, patients with less severe and less frequent attacks show a better therapeutic response than do those with more advanced coronary insufficiency. In our hands, aminophylline, because it is best tolerated by the digestive tract and does not require as large doses as some of the other members of the group, has proved the most satisfactory drug. It may be given in doses of 0.2 Gm. (3 grs.) three or four times daily. Enteric-coated tablets are available and may be prescribed for those who report digestive disturbances.

Recent studies made in our laboratory indicate that the duration of action of this drug, when taken by mouth, lasts only for a few hours. This observation suggests that perhaps smaller doses, taken at shorter intervals, might exert a more beneficial action. Work designed to indicate the most effective method of administration is now in progress. It is our present opinion that in certain cases the xanthines, because they dilate the coronary arteries, bring about symptomatic improvement. Whether, if taken for weeks or months, they aid in promoting a collateral circulation, cannot be decided on the basis of the evidence now at hand.

Surgical Procedures. Most patients with cardiac pain respond favorably, in some degree, to medical therapy. But there are those who suffer so intensely that they question the desirability of living. Fortunately, they do not form a large group. They welcome with enthusiasm any form of therapy which offers a reasonable hope of relief.

Of the various operative measures which have been suggested, only one will be considered at this time. Cervical sympathectomy and total

removal of the thyroid gland have not stood the test of trial and have been almost universally discarded. Section of the dorsal nerve roots, performed on a few selected cases, has been successful; but it is a major operation requiring laminectomy and presents a real hazard for the patient with advanced coronary disease. The operation for the formation of a new blood supply to the heart, as devised by Beck,[10, 11] holds out real promise, particularly if the technique can be simplified.

There remains for discussion paravertebral block with alcohol. This was first suggested by Swetlow in 1926[12] and has been done in various clinics, both in this country and abroad. The largest series, 62 cases, has been reported by J. C. White of Boston.[13] My own experience, recently published in collaboration with R. L. Moore, is based on observations in 45 patients.[14] Since our results and those at the Massachusetts General Hospital are comparable and similar, I will confine my comments to the material with which I am familiar.

Alcohol block is a simple technical maneuver; but it requires an operator familiar with the landmarks, skilled in their use and deft because of constant practice. The effectiveness of the injections depends entirely upon the accuracy with which the ganglia and rami communicantes are infiltrated. Our results may be summarized as follows:

Of 40 patients subjected to final analysis, 77.5 per cent experienced varying degrees of improvement; no relief was obtained in 22.5 per cent. In 47.5 per cent of the total number benefited, improvement was permanent to the time of the last follow-up examination, which in 2 cases was more than 9 years. In 40 per cent, relief was marked, in 5 per cent it was moderate and in 2.5 per cent it was slight. Temporary relief was obtained in 30 per cent. This was marked in 15 per cent, and moderate in 15 per cent. In short, there is a 75 per cent chance of relief; and if relief is obtained, there is an even chance that it will be permanent. Neither the duration of symptoms nor a previous history of coronary occlusion nor the degree of incapacity prior to injection appeared to influence the outcome. The postoperative appearance of Horner's syndrome was not essential to a successful result.

In none of our cases was relief complete. Even when a result was obtained which was satisfactory to the patient, some form of discomfort persisted, to act as a warning signal. There were sometimes actual twinges of pain, of less intensity than before injection, or a sense of pressure beneath the sternum. In several cases, mild dyspnea or a sinking

sensation in the epigastrium was substituted for pain.

Almost invariably (in 38 of 40 patients) painful intercostal neuritis was observed after the injection. This was severe in only 3 instances. It lasted from 2 weeks to 3 months but eventually always disappeared. Whereas it was a cause of complaint, the relief of cardiac pain, when obtained, more than compensated for discomfort. In 5 cases, a small pleural effusion developed which was always absorbed within a week. Pneumothorax, reported by others, was not observed. Increased temperatures, from 99 to 104° F., occurred in 25 patients; fever never lasted more than 4 days unless complicated by pleural effusion. There was no operative mortality.

Why, in some cases, relief should be temporary and in others permanent, has been explained by the recent experimental observations of Merrick.[15] He found that in cats, when a ganglion was infiltrated with alcohol, permanent block was produced to all regions innervated by the postganglionic fibers taking origin from it. This occurred because the alcohol killed the ganglion cells. When the rami communicantes alone were infiltrated, only a temporary block was produced; for regenerating fibers penetrated fairly early through the connective tissue scar formed at the site of injection. Partial block in patients is probably due to incomplete destruction of the ganglion cells or rami.

There is some reason to believe that abolishing or relieving cardiac pain not only affords symptomatic relief but has an effect on the coronary circulation and so on the heart muscle. A group of Toronto investigators,[16, 17] working with conscious dogs, observed that when the heart was denervated, the mortality following sudden occlusion of a coronary branch was materially less than in the intact animal. These experiments were regarded as indicating that, by preventing afferent impulses from reaching the vasoconstrictor center, reflex spasm of collateral coronary arteries was prevented. It was apparent also that sympathetic denervation rendered the myocardium less susceptible to the onset of ventricular fibrillation. Clinical and necropsy observations support those made in the physiological laboratory.[18] Of 376 patients who died of coronary sclerosis and thrombosis and were examined post mortem at the Presbyterian Hospital, 52 died suddenly. In the group with pain, death was sudden in 27.5 per cent, and in the group without pain in only 9.1 per cent. Regardless of the extent and character of the coronary lesions, the presence of pain tripled the incidence of sudden death.

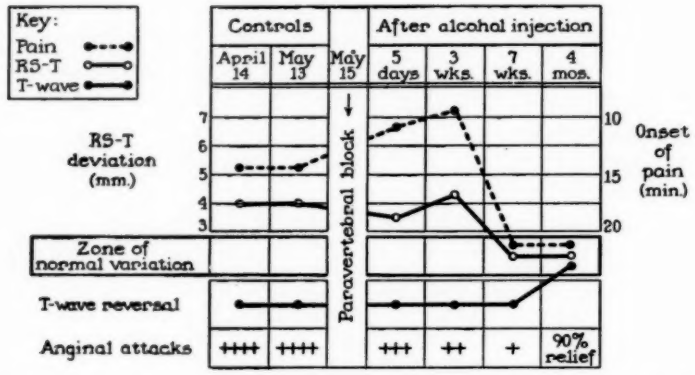

Fig. 2. *Case 2.* O. F., male, aged 53 years, automobile salesman. Anginal pain for five years; unable to work for the past three because of disability. Anoxemia test positive prior to paravertebral alcohol block. Four months after injection, coincident with clinical improvement, test became negative.

In 2 of our cases, changes in the form of the electrocardiogram occurred shortly after alcohol block. The alterations in the complexes were in the direction of normal and suggested an improvement in the coronary blood flow.[14] Observations with the aid of the "anoxemia test," made before and after injection, have shown in the one patient in which this has been done, remarkable improvement in the coronary reserve, coincident with clinical improvement (Fig. 2). A brief abstract of the clinical record follows.

Case II. O. F., aged 53 years, a male, automobile salesman, for 5 years had been having typical anginal pains radiating to the left arm. Disability became progressively more marked and for the past 3 years he was unable to work. He also had a feeling of substernal pressure and dyspnea after meals. He was unusually intelligent and coöperative.

Examination showed no cardiac enlargement, either on percussion or in the teleroentgenogram. The heart sounds were of fair quality. There was no gallop; a blowing systolic murmur was heard at the apex. The blood pressure was 140/82. The electrocardiogram showed regular sinus rhythm with a Q wave present in leads one and two. There was no defect in conduction and the T waves were normal. There was no anemia. The Kline test of the blood was negative.

The anoxemia test, done on a number of occasions, was positive.

Two controls, obtained at an interval of a month, were identical (Fig. 2). They show the occurrence of pain at the end of 14 minutes, a total RS-T deviation of 4 mm. and significant reversal in the direction of the T waves.

Paravertebral alcohol block was done on May 15, 1941 by Dr. R. L. Moore. Injections were made in the region of the first five dorsal ganglia. Horner's syndrome developed. There was moderate pain from intercostal neuritis, which disappeared in the course of 2 months. At the end of 4 months, he estimated the degree of improvement at 90 per cent. He was able to walk 2 or 3 miles without discomfort, except after a heavy meal. He had not used nitroglycerin since the operation and stated that dyspnea was gone. The blood pressure was 140/80. The form of the control electrocardiogram was unchanged. The anoxemia test at this time was negative.

In view of these various pieces of evidence, it appears that interruption of the sensory pathways, in certain cases, may diminish or abolish spasm in the coronary bed. This is a desirable effect, tending to promote a more adequate collateral flow in unaffected vessels. As has been previously suggested, earlier relief of pain in patients with coronary sclerosis might be expected to influence the course of the disease in a favorable manner. But in the more advanced cases, even when the result was good, unilateral alcohol block did not prevent the later occurrence of coronary occlusion or of sudden death.

In conclusion, I would like to leave with you this thought. The patient with recurring cardiac pain suffers usually from a progressive, disabling disease. Yet in some cases there is a remarkable and unexpected turn for the better; in others, the increase in the symptoms of coronary insufficiency is gradual and the degree of disability is slight. The physician can bring both aid and comfort. He is sometimes discouraged when improvement is slow and his therapeutic efforts appear to be ineffectual. He will find it helpful, under such circumstances, to keep in mind three master words—resourcefulness, patience and optimism. Of these, the most important to both doctor and patient is optimism.

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THE PSYCHOPATHOLOGY OF PSYCHOPATHIC PERSONALITIES*

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PSYCHOPATHIC personalities have been described in a variety of ways from the time of Koch[1] in 1893. He and various followers were impressed by the finding that there were many who though not having any specific mental illness were, nevertheless, not to be termed normal in their mental states. It was noted that these individuals had differences in one way or another from the ordinary everyday person-differences which set them somehow apart. Psychiatrists from the first tended to view those deviations as being congenital lacks and they included feeblemindedness as one of the types as well as hysteria and psychasthenia.

The psychic inferiorities were however subjected to further analysis as a result of which intellectual defects were withdrawn from this group as being a well defined entity which was susceptible of fairly sharp delineation. For the same reason Adolf Meyer[2] separated neurasthenia, psychasthenia and hysteria from the psychopathic states, preferring to keep the latter designation for a variegated assembly of what he termed "inferiorities not sufficiently differentiated." These he characterized as constitutional psychopathic inferiorities. The fullest descriptive accounts of the clinical pictures of these psychopathic personalities we owe to Grasset[3] in his book entitled "The Semi-Insane and the Semi-Responsible," and to Kraepelin[4] who in 1915 gave the classic delineation of seven types of deviated personalities: the excitable, the unstable, the impulsive, the eccentric, the liars and swindlers, the anti-social and the contentious.

Since that time the importance has been increasingly realized, both for the individual's life adaptation and for that of society in general, of the phenomena, the understanding, and the management of such prob-

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lems. Their wide range, their great numbers, the complexities involved in attempts to deal adequately with them, have led May[5] to declare that "the influence of these individuals on the community at large is something we have no means of estimating at the present time," and that there is no greater problem today than the attitude of the state towards the psychopathic criminal. White[6] stated the need for our attaining an understanding of these conditions in order that society may take the viewpoint of intelligent management and treatment rather than continue with a punitive and retaliatory attitude based upon misconceptions of the real nature of the psychopathic states.

When we seek to begin a survey of the situation with some clearcut definitions, difficulties at once present themselves. As recently as 1921, psychopathy was regarded by some as synonymous with the term insanity. Bleuler[7] has stated that it is but vaguely differentiated, that it deals with "deviations from the normal in all possible directions and mixtures." Kahn[8] tells us that it is impossible to give an exact definition of the psychopathic personality, so that we cannot say "Psychopathic personalities are——," but must be content to observe, "By psychopathic personalities we understand——." Kurt Schneider's[9] statement is an admirably restrained one. He says, "Psychopathic personalities are those abnormal personalities who suffer from their abnormality or from whose abnormality society suffers."

This places the accent upon the basis of community interest or upon the individual's own reactions to his condition. It is our present purpose rather to examine into the actual facts of this interesting mental deviation and to consider some of the details of its psychopathology, to determine whether any closer approximations can be made to a definition or clarification of the mental processes involved.

We may then review some of the more familiar symptomatology which clinical observation has amassed. While there is a great variety in the actual conduct of psychopaths, there are least nine aspects of their reactions some or all of which are so regularly found as to justify special attention. They are the following:

1. Inability to postpone
2. Ineffective consideration of consequences
3. Insufficient learning from experience
4. Faulty synthesis
5. Disproportionate responsiveness

6. Affective dominance over intellect
7. Disvaluation of reality
8. Disregard for truth
9. Insufficient social valuation

We will proceed to examine these factors. The first was an incapacity for delaying. It may be shown in the symptom of spendthriftiness, wherein one wants things and proceeds immediately to their purchase, if only the needed cash is in his pockets. Or it may appear when one repeatedly loses jobs because he flares up and voices resentment at his boss too outspokenly. The issue here seems to be that the long circuiting characteristic of our more complicated reflex responses has not taken place; the stimulus has led too directly to an outflow in motor behavior. Such individuals cannot sufficiently recognize that a gratification dependent on delays might mean more than a trivial appeasement at the instant. Apparently the craving for some instinct satisfaction exerts too much pressure upon the psychopathic individual.

Related to this difficulty is the second one of a failure to give adequate consideration to consequences. An act by itself might seem justified but when we realize what may result from it, that future possibility often acts as an effective deterrent. This faculty of mind serves to bring the future into the realm of present considerations. Not to be able to give due weight to possible consequences means that one has not sufficiently organized the material of the future. In the complexities of modern living it is more necessary than ever before to keep forming incessantly new estimates and forecasts of the probabilities to come as well as they can be seen from the implications of the present. This however the psychopath often does not do. He will, for example, invent the most fabulous lies without stopping to realize how deeply they will inevitably involve him tomorrow. Or he may sign leases, enter into business contracts, execute promissory notes without being impressed by the need to recognize the demands and the means for ultimate fulfillments.

Each individual must make such blunders in the process of learning these needs and developing skills in meeting them. When the forecast indicates that they cannot suitably be met he calls upon the past to be reminded of some better course to follow. The psychopath, however, does not learn effectively from even repeated experiences. If he cannot organize his future, here is an indication of his equal inability to make adequate use of the past. It seems that he is not able to formulate general

principles from the experiences of his living. Their type value is apparently of less significance than their individual instance value. This is a corollary of his tendency to live in the present with overvaluation of the immediate moment. He cannot detach himself sufficiently from his intimate contact with the single items of his living to view them impersonally or objectively enough to derive the rules which they evidence. People with psychopathic personalities not infrequently have a remarkably accurate capacity for critical self evaluation. They are, however, never able to make use of this verbal insight. And their most fervent resolutions for improvement, although eloquently and correctly reasoned, invariably fail.

This is perhaps due to the fourth item under consideration, that of the psychopath's tendency to make incomplete or faulty synthesis. Part of his difficulty in learning the lesson of experience comes from his propensity for dealing with part pictures rather than with the total picture of his situation. The lack of synthesizing ability is probably due to the immediacy in response. The psychopath makes his reaction often to the first portion of a situation which comes to his notice. If the idea of marriage occurs to him, he may propose and be accepted before the financial implications of the marriage have reached his consciousness. Another will make a suicidal attempt because he has just been reprimanded, failing to form any conception of thus receiving training which may help him earn a larger salary. This tendency is readily recognizable in an immature, growing child. It seems in the psychopath, however, to be a more erratic and variable trait, now present, now absent. Like other psychopathic traits it shifts whimsically, being manifested to a differing degree from topic to topic or from one time to another.

A fifth factor equally unpredictable and whimsical is that of unbalanced and changing intensity of emotional response. The customary variabilities of affective coloring show a certain consistency, a roughly similar quantity of affect usually accompanying a given phenomenon. The psychopath, however, seems to have in his bewildering and shifting affectivity a set of emotional values for the items concerned which is quite different from that of the average individual. Thus, the most well-meant bit of teasing may produce a violent rage or a flood of tears, or a word of routine praise may raise one to the skies. A small disappointment can convince a psychopath of the necessity of suicide or a casual remark may suffice to start him zealously upon some previously unconsidered

life vocation. If the emotional accenting within the self is eccentric or disproportionate, likewise its intensity of expression may show gross difficulties of control. "I know it is silly but I cannot help feeling that way," a psychopath will say.

Accordingly it is understandable that the psychopath's intellectual functions may be dominated by his emotional stresses. It seems, indeed, that this is not only true in many instances but that it is a general phenomenon in such persons. It is one of the regularly occurring evidences of immaturity, that the force of emotional argument looms greater than any factual intellectual construction. Wishful thinking is nowhere more in evidence than in this group. One decides that the world owes him a living. He is convinced all evidence to the contrary that he is vastly superior to his fellow men. Never managing to sell one of his productions will not prevent his deep certainty that he is a supreme poet or novelist. We deal here with another approach to several of the factors previously mentioned and indeed all of them are inextricably interrelated. In a given instance it may be impossible to decide which may be causative in producing the other. Such a consideration suggests that there may be some formulation underlying the various factors which might explain their coexistence and their interdependence.

The psychopathic personalities are not psychoses and hence it is not to be expected that reality values of the external world should be actively denied by the psychopath. This is indeed borne out by clinical observations. Reality is not, however, permitted to have its full significance. If he deals with incomplete syntheses not making adequate use of past experience and suffering emotional warpings of his judgment faculties, the psychopath is certainly not making correct, adequate use of reality. He cannot evaluate reality in its true significance when he is aware of it only in fragments. It may therefore be said that it is a trait of the psychopath to disvalue reality rather than to deny it. This may be illustrated for example by the fifty-year-old able-bodied man who demands money from his feeble, aged mother "because you are my mother and should therefore support me." The true significance of his circumstances, his actual responsibilities for failures, crimes or disappointments, may all be glibly talked out of existence.

That this might indicate insincerity in one who really knows how the facts stand merely puts the shoe on the other foot, for he then shows a tendency to misrepresent things and evidences a disregard for truth in

its own right. The incontrovertible majesty of the existence of fact remains for most persons quite indisputable. To the psychopath this frequently is not so, as is particularly striking in the case of pathological liars. Their distortion of truth often amazes even themselves and they are at a loss to explain their motivations.

The last point to be itemized lies close to the one just stated, but in our social culture it has attained such preëminent importance as to justify its individual mention. One of the strongest influences on the cultural advance of civilization has been the establishment of such social forces as morals, traditions and precepts, and the formulation of such conceptions as those of duty, reputation, honor and pride. These are psychic processes which are included in the Freudian concept of the superego. To them we owe the increasing effectiveness of management and control of id and ego tendencies. But it is just here that the psychopath is likely to show particular weaknesses and inabilities to measure up to the customary attitudes of his fellows. The call of duty may be entirely overcome by inherent laziness; the measure of personal pride is often obliterated by the demands of less valued impulses; and reputation, though desired, may not produce enough sustained effort to be attained. Again we face a problem of evaluation in which continuity of influence seems less to the psychopath than to the average individual. A social force is readily overbalanced by a personal inclination. Apparently the cultural values are viewed from behind the psychopath's personal ramparts and are not taken into himself to become part of his own personality, there to cope with other traits in his own makeup.

The result is that the psychopath is more self-centered than the ordinary individual, and that his mental processes appear to behave in a less mature fashion than we expect from the ordinary person. It may be inquired if such traits are inherently different from those of other people, or if they are maladjustments of machinery which if properly tuned should be functioning properly? The importance of such a query is apparent for a rational approach to therapy.

There has not been reported any structural pathology which would offer a somatic or neuropathological foundation for psychopathy. Hence we must consider it as a functional abnormality, a behavioral deviation from what is customary. As soon as that step has been taken, we must seek explanation of the phenomena in terms of dynamics. When we consider a psychopathic personality from this aspect, we have a problem

which calls for analysis of the mental functions themselves. Let us look therefore more in detail at some of those nine types of psychopathic symptoms which have been referred to above.

One was an unsuitable immediacy. Why "unsuitable?" Because our usual ontogenetic development takes us beyond that level of reactivity, so that it represents a functional deviation from the group, a sort of deficiency. We assume that an individual confronted simultaneously with various issues will ordinarily delay action, that is, he will delay in adopting a finally decisive response pattern until, through the processes of association, he has found the most effective compromise he can. This resultant indicates the relative importance he recognizes, according to his own personal experience, in each of the issues he thinks are involved. From his methods of response we can infer, if the various issues are known, a good deal about the proportionate weight he feels each issue carries.

The indecision of a psychasthenic keeps him for hours from selecting between two neckties, and we consider this an evidence that values are quite evenly balanced. The psychopath's undue speed in settling his decisions approaches the other extreme at which one factor is not only given preference, but exclusive or preponderating preferment when other issues should have their real values also under consideration. If certain details preëmpt his attention, others can exert only a lesser claim upon the psychopath for his consideration. It is not our present purpose to determine any ultimate reason for this but merely to call to attention that it does occur in psychopathic reactions. This makes it appear that one factor in the psychopathic phenomenology may be an overvaluation of certain material so that it acquires an improper ascendency over other items. It would not matter what the overvalued items were but rather that some overvaluation had resulted in a corresponding undervaluation of other material so that the response lacked in validity, in adequacy, in accuracy; or, we may say, in general acceptability.

If others of the psychopathic qualities mentioned are examined, it will be seen that they too contain this same difficulty. When the psychopath fails to take due heed for consequences, he is living in the present or near future without seeing the somewhat more distant future as clearly; without granting to eventualities as much importance, as others do. In other words the meaning or interest of the present is still as it was with all of us in childhood, more vivid and more real than is that of a dim,

vague future. Once more we see that certain items are still holding first place in the psychopath's mental functioning which should have made allowance for the coexistence and the significance of other items. That these latter seem vague, or less significant, is but another way of saying that one's accented attention remains still upon the former items. This is to say, that lack of diffusion of interest has caused a continuing fixation which is inadequate because it takes into account too limited a portion of reality.

It is the same with the classical incapacity of the psychopath to learn some of the vital lessons even through repeated experiences which contain the material that might teach him. It is a common clinical observation that, although he may be able to state in glib exactness all sorts of precepts and maxims which are logically derivable from his experiences and those of others, the psychopath seems not to be actually touched by the meanings inherent in his words. Even though his intellect has worked out the answers, they have not acquired enough comparative affective weight to claim parity with his overly egocentric notions. If one cannot be influenced by the book he will not read, no more can he evidence deep-rooted effects from his own experiences if he has not bestowed sufficient attention upon them to make them glow with a personal, meaningful value. "Yes, quite true," he will say, "but what does that mean to me? Nothing."

The other types of psychopathic symptomatology can be viewed from the same standpoint. The disvaluation of reality, for example, is something quite different from a mere honest difference of opinion as to the significance of facts, just as it is inherently different from psychopathic denial or delusional distortion of reality. What the psychopath does with portions of reality is to have their meaningfulness to himself as emotionally colored stimuli differ from what it is to the average individual. Hence the power of reality to move the psychopath, to arouse him, to teach him, to limit him, is not what it is to others. It may well be that some factor within himself is functioning in a way to lessen the meaning of outer reality to the psychopath, and this by the mechanism of no active inhibition or repression but through the more placid phenomena of mere limitation of the attention to details of more interest to him.

Perhaps we may learn from the popular social attitude which so often pronounces accusingly about some dereliction: "If you cared enough

to have thought about it, you would have realized." Evidently the psychopath does not "care enough" about many of the items and the issues which have considerable meaning for most of us. And as he obviously has no disinterest, but rather too intense an emotionality, it would seem in line with clinical facts that his interests are simply centered elsewhere.

The majority of individuals, through experience and training, develop a certain degree of social-mindedness in which their sense of values is largely predicated upon social consciousness; their decisions are modified by regarding themselves as an inherent part of the group. Their affects and behavior are thought of as modifying and as being modified by those of others, and hence are regarded by those individuals as requiring frequent checkups so as to be kept, like one's watch, set correctly with others. The psychopath on the other hand has to those individuals something of the same comparison as had the Ptolemaic theory to the more advanced Copernican theory of the universe. To the psychopath he himself is the center of the universe about which everything else circles. In his essentially narcissistic viewpoint, whatever is outside of the focal point, himself, is tacitly excluded; it is not absorbed as being a part of his own self. His boundaries are quite personally limited. From the social approach this means that he lacks in that social identification which would properly articulate his inner self with his associates. From the standpoint of personal psychology his concerns seem to have been fixated upon pure self interests which characterize mere egocentricity.

It must be asked: If the psychopath is a narcissist, if he is so absorbed with the fascination of his own inner drives to the detriment of the usual wide derivatives which lead others to augmented horizons and to broadened, socially controlled interests in life, then, how is his state different from that of dementia praecox, which is likewise termed a narcissistic state? A distinction can be drawn as follows: Dementia praecox produces actual distortions of outer reality, as by believing the hospital nurse to be one's deceased grandfather. Psychopathic personalities do not make such a distortion of factual realities, but they have a personal set of values for external realities so that to them those realities hold different implications, leading to non-usual associations or behavior. Thus, a schizophrenic planning his day's work may be interrupted by hearing God's voice; but the psychopath, by being so intrigued with a discussion of poetry that the conceptions of duty, punctuality, reputa-

tion and personal pride in achievement confront him with comparatively lesser urgency.

The psychopath is susceptible to the push of externally applied motivation, but often seems not to have enough energy available to motivate himself from within in the way we say he should. But if the pressure implicit in the words "should" or "obligation" or "expected" which come to an individual from outside were withdrawn, he would be left with only the impulsions from his inner self. These, if considered at an early period in life, are utterly egocentric; they make use of externals for personal gratification alone. So it seems to be with the psychopath, who to varying degrees regards the world of people and situations about him, not so much in their own right as existing entities themselves, but rather in relation to their potential adequacy for gratifying his own immediate wishes.

Here we return to a factor referred to above. The psychopath seems to have a responsive tendency which is uneven, perhaps explosive, at any rate often disproportionate. This is because we judge of reactions in their relationship to the outer world of reality. We expect similar settings of the external picture to call for similar graduations of an individual's inner responsiveness. When the external situation is, say, seven o'clock, and Monday morning, and job duty in one hour's time, our regard for the pressure of the external facts makes us get out of bed. But if one's mental associations are restricted to the planning of a party for that night, the pressure towards arising will be markedly decreased. Such an inner primacy of wish-phantasy-urges is the dominant factor in the psychopathic personality. That is why his responses may seem out of adequate proportion. His inmost balances of affectivity and the instinct drive-urges of the moment are what determine the outflow of his energies, rather than the stage setting in which he finds himself and by which standard we are accustomed to measure his adequacy.

To sum up, it is the view here set forth that in psychopathic personalities there is to be found as a definitive characteristic a certain fixation or limitation in psychic development. This consists of a continuing restriction of affective attachments such that significance or urgent motivating value does not widen its scope to include as much of the world of reality external to the self as is usual in adult life. It is a permanence of some attitudes of human psychology which were normal for one level of the evolving mental complexity, but whose continuance is indica-

tive of a form of persisting immaturity. It is an enduring tendency to measure by a personal rather than by a social or group yardstick, but involving no further distortion of reality.

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THE INFLUENCE OF CLIMATE AND GEOGRAPHY ON HEALTH*

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IT IS with deep appreciation of the honor conferred upon me in being asked to deliver this Lecture that I speak tonight. Particularly appropriate is it that the title of the lecture suggested by your Committee on Public Health Relations specified the influence of climate and geography on *health* and not on *disease*. Factors controlling health and well-being are always more important, because the greater part of any population mass is composed of people who are in good health and who wish to remain in that favorable state. Analysis and elucidation of these factors underlying maintenance of health, however, are greatly facilitated by close scrutiny of conditions predisposing to disease. In the presentation tonight I shall therefore set forth a picture of climatic and geographic dominance over health much more extensive in its ramifications and implications than is generally considered, but much of this picture will be based upon the study of climatic and geographic variations in certain types of disease. The geographic factor will be disregarded except as climatic characteristics vary from one region to another. Certain transmissible diseases show a geographic influence quite divorced from climatic effects, dependent more upon density of population, urbanization, industrialization, freedom of travel and intercourse, and upon a variety of other such factors not to be considered in the presentation tonight. For the sake of clarity and emphasis, attention will now be directed almost exclusively to the climatic factors affecting the maintenance of health.

In considering these climatic factors it is necessary to look upon man (and other warm-blooded animals) as an energy conversion machine—a machine that operates only by virtue of the energy it derives from the

* The Hermann M. Biggs Memorial Lecture delivered before The New York Academy of Medicine at the Stated Meeting, April 4, 1941. Illustrations used at the time of delivery have been omitted from the text here published. Most of them may be found in the author's book "Medical Climatology," published by Charles C. Thomas, Springfield, Illinois, 1939.

cellular combustion of foodstuffs. An individual is alive only so long as this combustion continues, and the level of his vitality seems determined by the intensity of his tissue fires. He is not an efficient energy-conversion machine, however, being able to utilize for work performance only 20-25 per cent of his total combustion energy. This level of working efficiency is about the same as that of a good gasoline motor, but is far below the 37 per cent efficiency obtained from Diesel engines. Other warm-blooded animals (horse, dog) exhibit this same 20-25 per cent level of working efficiency.

The human body in action is therefore faced constantly with the necessity of dissipating into its surroundings the remaining 75-80 per cent of its combustion energy as waste heat. It must be able to dissipate this heat readily, for any heat accumulation means fever and severe disturbance of body functions. The human body is much more sensitive to over-heating than is any inanimate motor and so has developed an intricate and complicated heat loss mechanism involving vasomotor control of blood supply to the skin and activity of the sweat glands. This heat-loss mechanism operates efficiently to take care of the short-term loads that go with periods of bodily activity or sudden difficulty in heat loss brought about by a rise in external temperatures. It is not necessary to consider here the operating details of this heat-loss mechanism beyond saying that its adaptability is truly remarkable and its capacity great indeed.

When the body is faced with prolonged difficulty in getting rid of its waste heat, however, it tends to bank its tissue fires and reduce the load upon this delicate heat loss mechanism. Difficulty in heat loss continuing for only a few days results in little or no adaptive change of combustion rate, but, if it persists for 10 days to 2 weeks or more, then there occurs a definite slowing down of cellular combustion and a more sluggish pace of life. The belief has for some time prevailed in American medical circles that man shows no such seasonal response to changing temperature level (this is discussed in a separate article, *Amer. Jour. Hyg.*, May 1939, *29*, 147-164). Careful review of the literature and the evidence available, however, indicates clearly that men do indeed show this responsiveness to change in external temperature level. This is an important point, for the general vitality and existence level of individuals now seem determined by tissue combustion level and any factors altering the tissue combustion level at once assume great importance in

life. The major effects of climate upon human existence become more easily understood when we view them against this background of climatic dominance over ease of body heat loss.

Difficulty in the dissipation of the body's waste heat, prolonged beyond two weeks, results in a slowing down of tissue combustion, and with this goes slower growth, retarded development, reduced fertility, lessened resistance to infection, and lowered energy for thought or action. Under conditions where optimal ease of heat loss prevails, on the other hand, growth is most rapid, development of sex functions occurs earliest, fertility is highest, ability to produce immune bodies and to resist infection greatest, and energy most abundant for the support of an active existence. Body form is distinctly more rotund in stimulating coolness and general vitality higher by every measurable index.

Human growth in tropical warmth is retarded just as is that of experimental animals, while in middle temperate latitudes it proceeds most lustily. During a very recent stay in Panama the author had an opportunity to study height-weight data on Canal Zone school children, and to see what effect varying periods of residence in the tropics have had upon their growth. The Canal Zone offers a most valuable opportunity to study climatic effects divorced from those of tropical disease and faulty diet. Zone sanitation is on as high a level as that of the most progressive northern cities, while the bulk of the food is imported from the United States. Meats sold in the "gold" commissaries are imported from the United States or Argentina. American children of any age, born on the Isthmus, are considerably taller and heavier than are native Panamanians; but those born in the Zone are inferior in stature to those newly arriving from the United States during the recent influx for new lock construction.

Most significant is it, however, that the stature of those children born in the United States was found to deteriorate more and more with each additional year of residence in the Zone. Here is almost pure evidence of climate working alone, evidence pointing to the same depressing effects of tropical warmth as were observed in experimental animals.

A sharp lowering of human reproductivity takes place during seasons of depressive moist heat at southern latitudes or during seasons of exhausting cold in the north. Human fertility rises highest in any locality when mean monthly temperatures are around 65° F, and is depressed when winter temperatures fall below 40° F or those of summer

rise above 70° F. Human conception rates may be suppressed as much as 50 per cent by severe summer heat waves that are prolonged beyond a duration of two weeks.

The onset of menstrual cycles in girls also shows the same retardation in tropical heat that is shown by sexual development in experimental animals. Menarche comes later in tropical girls, and not earlier as is so universally believed by both medical and lay persons of all countries. The widespread belief in early tropical maturity is found expressed in medical literature back through the centuries to the time of Hippocrates in Early Greece, but always unsupported by any statistics that may be available. It is a belief that may well have been handed down through several thousands of years since the time when even present middle temperate regions had polar climates and optimal conditions for man existed only in what are now subtropical lands. Certainly at present girls mature earliest in stimulating middle temperate lands, with the menses beginning 1 to 2 years later in either depressing tropical warmth or exhausting polar cold.

The onset of fertility is also delayed most in those regions where the menses begin latest. Although sexual intercourse goes on quite uninhibited in tropical warmth, illegitimate first births come at practically the same maternal age as do the legitimate. Among people of middle temperate climates, on the other hand, illegitimate first births come at much earlier maternal ages. All phases of development are thus accelerated in cool regions—the menses begin earlier, fertility comes on sooner, and the period of adolescent sterility is shorter than in tropical warmth (see *Human Biology*, 1936, *8*, 607, and 1937, *9*, 43).

In ability to resist infectious invasion and to produce immune bodies against invading organisms, men and animals are again found more highly vital when proper ease of body heat loss permits them to maintain an active tissue combustion. Depressing moist warmth (for even as short a period as 3 weeks) produces a sharp reduction in the ability of white mice to survive inoculation with a virulent pneumococcus organism. Even though mice be healthy and on a completely adequate diet, those adapted to a 90-91° F environment are practically all dead from a pneumococcic inoculation before those at 65° F begin to succumb. Production of immune bodies after vaccine administration has been found much more active at 65° F than at 90-91° F, both for pneumococcus vaccine in mice and for typhoid vaccine in rabbits.

Certain human infectious diseases show this same relationship between survival rate and ease of body heat loss. Among some 3100 tuberculosis deaths of indigent patients in a large sanatorium near Cincinnati it was found that those patients born in Cincinnati or farther northward survived the ravages of the disease twice as long as did those born in states bordering the Gulf of Mexico. Actual survival times, from first symptom to death, were 22.7 months for northern-born whites and 11.8 for the southern-born, 16.8 for northern-born negroes and only 9 months for those who had migrated up from the Gulf states. Among European migrants, the survival time was 20.6 months for those from Western and North Central Europe but only 11.7 months for those coming to Cincinnati from Mediterranean countries. Acute appendicitis also shows a markedly higher fatality rate per 100 attacks in the south than in more invigorating northern latitudes. Nor does the more favorable prognosis in the north necessarily reflect better surgical skill or more competent care than is available in the south, for Boston's fatality rate is higher than is that of less favored cities in the upper plains states or provinces. Acute nephritis is another acute infectious disease showing highest death rates in tropical or subtropical warmth and lowest in middle temperate coolness.

In general it is infectious disease that kills off people in the tropics, while in cooler temperate regions death comes more from diseases of degeneration and metabolic breakdown (arteriosclerosis and heart failure, cancer, diabetes, etc.). Laboratory animals kept free from infectious hazards in a warm environment live much longer than do those forced into a more rapid pace of life by stimulating coolness. But the average life span of man in the tropics is shorter, rather than longer, because of the lowered resistance to infection and the greater prevalence of pathogenic organisms.

In certain regions of the earth climatic stimulation rises high and drives man impetuously forward into a highly vital and energized existence. In the broad expanses of tropical lowlands, however, man is held down close to a vegetative level by the difficulty he must always face in getting rid of the waste heat that would be generated by a more active life. Year after year and for centuries without end tropical man is smothered under this blanket of moist heat that saps his vitality and keeps him in a low estate. The racial importance of this climatic depression takes on sharper emphasis when it is realized that over half of the

earth's human population lives under this severe physical handicap. Only in a few favored regions of the earth are climatic conditions such that man can expand and reach the full flower of his developmental possibilities. We here in North America enjoy one of the largest and best endowed of these energizing regions. Southern Australia, New Zealand, and West Central Europe are almost as well favored. Certain limited plateau areas within the tropics are lifted out of the tropical depression by virtue of the altitude factor.

Energizing climates are not unmixed blessings, however, for man does not seem to possess unlimited ability to respond safely to increasing climatic stimulation. In the most stimulating climates of the earth he is showing alarming evidences of bodily and mental breakdown from the fast pace of life. These evidences of stress are most severe in those parts of the body economy most intimately concerned in the combustion processes. Perhaps 85 per cent of the energy used in living is derived from cellular combustion of glucose. It is not surprising, therefore, that breakdown in the body's ability to mobilize and prepare this sugar for cellular use should occur most frequently in populations living where tissue combustion is most active. Diabetes mellitus represents just such a breakdown in the body's ability to transform the food eaten into the special form of glucose needed for cellular combustion. Diabetic death rates rise highest in urban populations across middle temperate latitudes of America, but there is also a ten-fold rise in colored rural death rates from the disease from Gulf states northward to Michigan and New York. Diabetes exists in tropical countries, but largely as a non-troublesome glycosuria with little tendency for the development of acidosis or coma. In more stimulating climates, however, the disease is severe and the dangers of acidosis and coma very real.

Another way in which the stress of too energizing a climate is manifested is in exhaustion of the body tissues producing the red blood cells. Only in the most stimulating climates does pernicious anemia assume troublesome proportions. Its death rate rises highest in those same middle temperate latitudes of America where diabetes death rates are highest. Death rates for toxic goiter, leukemia and Addison's disease show similar climatic relationships.

Most striking, however, is the close relationship heart failure shows to climatic stimulation and ease of body heat loss. Classification of vascular and heart failure deaths is still too confused and lacking in unifor-

mity from one part of the country to another for valid regional comparisons to be made. Seasonal swings in mean temperature level in a given region, however, bring just as marked changes in tissue combustion rate and load on the heart for oxygen transportation as do regional climatic differences. There has been shown a marked inverse relationship between heart failure admissions to the Cincinnati General Hospital and mean monthly temperatures throughout the year (dealing only with those heart failures not precipitated or complicated with acute infectious attacks or other direct precipitating events). These non-infectious heart failures are about four times as frequent in winter cold as in summer warmth. During certain Cincinnati winters when warm, balmy weather largely prevailed (1929-30, 1930-31, 1931-32) this type of heart failure dropped almost to the low summer level of frequency. This exemplifies clearly the changes in heart load and circulatory stress that accompany environmental variations in the ease of body heat loss. Circulatory load and blood pressures rise with winter cold and fall during summer warmth. Vascular spasm may add considerably to the circulatory embarrassment of winter weather.

There now seems little doubt that one of the chief factors activating or depressing human existence is mean temperature level and the ease with which body heat can be lost. With the active combustion of a cool region goes a higher vitality and more vigorous development along all lines. Even mental processes show this activation, for intelligence or aptitude tests given college students result in markedly lower ratings with tests given during summer heat. But certain hazards attend too active a pace of life, and it is this type of degenerative and breakdown disease which is now causing chief concern in the earth's most stimulating regions.

A second climatic factor, with effects upon human health quite different from those of mean temperature level, is cyclonic storminess. Sudden storm changes in the weather seem largely responsible for the timing and initiation of many types of infectious disease, particularly those of respiratory and rheumatic types. There occurs a marked increase in disability from respiratory infections among employed workers during winter storminess. Rheumatic fever attacks show a similar seasonal variation in frequency.

Temperate zone storminess is largely determined by the passage of alternating major "high" and "low" areas across a given region, bring-

ing marked and often abrupt changes in temperature, barometric pressure, wind velocity and precipitation. Each storm area produces pressure and temperature changes for hundreds of miles on each side of its center as it moves across the continent, so that people living in the great central trough of North America must face almost constant weather change during the winter season. In summer the storms are reduced in number and follow a more northerly course, leaving the South then under a blanket of almost undisturbed tropical moist heat. The Southwest (New Mexico, Arizona, and Southern California) is the only part of the United States unaffected by cyclonic storminess, and that is the region to which chronic sufferers from respiratory and rheumatic infections should go for refuge.

During certain years of unseasonable warmth winter storminess is greatly lessened, with almost as much of a reduction from normal as the summer decline from the winter level. During warmer and less stormy years respiratory disease frequency drops and the general health of the population shows marked improvement. Death and sickness rates rise during the colder, more stormy years. In the southern hemisphere, storm changes tend to be more evenly distributed throughout the year or even to be somewhat greater in summer than in winter. Thus winter existence down there does not carry the sharp increase in health hazards people face in north temperate lands where winter cold and the season of greatest storminess coincide.

Little information is yet available as to just how weather changes affect man. Simultaneous occurrence of changes in the several weather factors makes it difficult to unravel and study the separate effects of each factor. Some of the effects of sudden temperature change and body chilling or overheating are known, and some hints have been found that storm changes in barometric pressure may produce profound disturbance in body functions through alterations in the tissue water balance. However, the matter needs thorough study under controlled laboratory conditions where each factor can be investigated separately. Lack of laboratory facilities for such study has left us with only vague and fragmentary information as to just what effects weather changes do produce in the body. Findings of great health value will probably result when such an investigation can be properly carried out. It seems likely that sudden weather change will be found a potent factor in the *initiation* of many types of acute infectious attacks (particularly respira-

tory and rheumatic infections), but that *ability to survive* the infection depends more upon prevailing mean temperature levels and ease of body heat loss. Thus man has less respiratory infection in summer warmth, not because of a higher vitality during that season, but because of reduced storminess. Actually his ability to fight infectious invasion is sharply reduced in severe summer heat.

Let us now consider climatic effects in still another light, again involving the cellular combustion processes that furnish us the energy upon which we live. This time the vitamins are involved, particularly the B fractions, each of which now seems to act as a catalyst at a given stage of the glucose combustion. Intake requirements for thiamin and other members of the B complex have been found to be fairly constant in man and other mammalian forms under normal conditions. But if cellular combustion is slowed down by external warmth, then the vitamin required for handling each gram of food rises sharply. Twice as much thiamin per gram of food is needed by animals living in a 90° F environment than by those kept at 65° F. Foods with vitamin contents that are entirely adequate in a cool environment become sharply deficient in depressing moist warmth.

This has been clearly shown for thiamin requirements in rats. Food consumption and growth rate at 65° F are almost as good at 0.6 milligrams of thiamin per kilo of food as at any higher level, while at 90-91° F twice this level is required for optimal response. At 65° F real inadequacy is present at 0.2 and 0.4 milligrams per kilo of food, while at 90-91° F even 0.8 milligrams is clearly suboptimal. Preliminary studies have indicated that just as sharp differences will be found for pantothenic acid requirements and perhaps for pyridoxine. Others of the B fractions have not yet been investigated in this fashion.

Recent preliminary studies in Panama have convinced the author that this higher vitamin requirement in external warmth constitutes a severe handicap for people living in tropical heat. Not only are their B requirements higher because of the difficulty in heat loss and more sluggish metabolism, but tropically produced foods seem much more deficient in the B vitamins than are those of middle temperate regions. Meat animals grow much more slowly in the tropics, and their flesh is itself deficient in the B fractions. Eggs and milk products are also deficient unless the hens and cows are fed on vitamin-rich imported foods. It takes about 5 years in the tropics to produce a 1000-pound

steer which in temperate coolness can be ready for slaughter at 1½ to 2 years. A 200-pound hog is produced in Panama in 12 to 15 months but in northern United States in 6 to 7 months!

Tropical residents therefore face a double handicap with respect to their vitamin intake. Their needs are higher than in cooler lands, but their available supply is lower. Herein probably lies one basis for the poor nutrition and low vitality so prevalent among tropical residents. It should be borne in mind, however, that the most perfect diet cannot overcome all the handicaps of difficulty in heat loss such populations must continue to face. A higher and more healthful existence level might be attained by proper vitamin intake, but even the most perfect dietary would still leave them considerably below the high level of vitality and tissue combustion rate attained in middle temperate regions.

It is probable that this elevated vitamin requirement in tropical heat, coupled with the lowered vitamin content of the foods, is largely responsible for the tropical and subtropical distribution of such deficiency diseases as beri beri and pellagra. The matter needs close study in order that a more accurate evaluation may be made of these climatic differences in vitamin requirements and food deficiencies. Man faces enough of a tropical handicap in having to live under the depressing blanket of moist heat without adding to his difficulties by an inadequate vitamin intake.

It seems desirable to interject at this point some very recent observations regarding thiamin toxicity. Usage of this vitamin has reached very large proportions during the last two years. No reports of toxic reactions have appeared in medical literature and the dosages administered to patients have been sharply increased. Administration of 20 to 50 milligrams daily has become rather common practice, with some clinicians at times recommending as much as 100 milligrams and a few even 500 milligrams a day. No studies have been reported showing any clear advantage to be gained by dosages so far above the normal 2 to 3 milligrams daily need. Their only justification would therefore reside in unquestioned non-toxicity of the product. Such lack of toxicity seems quite fallacious, in view of observations now to be reported for the first time.

In August, 1940, the author first observed a toxic reaction in an otherwise normal Cincinnati resident after the daily ingestion of 10 milligrams of thiamin for 2½ weeks. The symptoms were those of in-

somnia, increased irritability, severe headache, rapid pulse, tremor, weakness and collapse. Recovery followed promptly after cessation of thiamin intake. After a week's rest, thiamin administration was renewed at 5 milligrams daily, and the same toxic syndrome recurred after 4½ weeks at this intake level. Recovery was again prompt after cessation of thiamin intake. While studying the thiamin status of people in Panama during February and March of 1941, the author saw many further evidences of severe toxicity in patients taking 10 to 50 milligrams daily as a more or less routine treatment to obviate the tropical letdown so commonly seen in migrants from cooler climates. The toxicity symptoms were similar to those of hyperthyroidism: insomnia, increased nervousness, hyperirritability, emotional instability, rapid pulse, palpitation, and in many cases nausea and vomiting. Prompt subsidence of symptoms followed cessation of thiamin administration.

Since returning to Cincinnati, the author has obtained evidence of even more severe toxicity, with collapse, syncope, circulatory shock and near approach to death.* These severe reactions have taken place at thiamin dosages of 20 to 50 milligrams continued for two weeks or more. It seems likely that many such severe reactions, and those of a milder character, will be reported now that attention has been drawn to the form such toxic response may take. Better control of thiamin dosage by excretion studies seems badly needed, in order that patients may have the benefit of as much of the vitamin as they really can use, but without being subjected to the hazards of unnecessarily high dosages.

It is possible that toxic reactions to thiamin occur more freely in people suffering from multiple B-fraction deficiencies, as suggested by Morgan in a recent issue of *Science* (1941, *93*, 261) and that this accounts for the prevalence of toxic reactions in the tropics where multiple deficiencies seem more prone to occur. The matter needs careful study at an early date if widespread occurrence of ill effects are to be obviated.

The finding of thiamin toxicity has no bearing upon recent steps being taken to fortify white flour or other foods with the vitamins they have lost in processing. No evidence exists to show that thiamin in normal physiologic amounts can be in any way harmful or toxic. Plans for fortification of foods entail only replacement of vitamins previously lost in processing procedures. The finding of definite toxicity from overdosage with thiamin does, however, necessitate a more cautious

* One death from thiamin injection has just been reported to me (May, 1941).

and conservative attitude from both medical profession and laity toward uncontrolled intake of these useful vitamin preparations as they become so freely available.

Health assumes then a much more dynamic and buoyant quality in the cooler regions of the earth where body heat can be easily dissipated. Even the vitamin content and quality of foods grown in such regions favor a more active and highly vital existence. In regions of moist warmth, on the other hand, man faces various types of handicaps. He must live at a slower pace because of difficulty in dissipating the larger amount of waste heat that would be entailed by a more active existence. His efficiency seems further lowered by a widespread dietary deficiency for the B vitamins in his foods coupled with a higher intake need. This broad view of climatic dominance over the dynamics of human existence offers a most useful basis from which to attempt an explanation of how climatic environment affects man's health and general welfare. Man is not a standard being of fixed characteristics, but is rather an energy conversion machine highly responsive to the physical and chemical influences affecting his ability to burn food and produce the energy whereby he lives.

Such a dynamic view of human existence at once opens up new possibilities in seeking a better adaptation of population masses to their environment and living conditions. It gives also a clearer insight into the human variations so evident in different climatic regions of the earth, pointing out the factors that hold man back in one land and push him irresistably onward into an active existence under more favorable conditions. It explains the sharp variations in rate of body breakdown so evident over the earth, and affords at least a partial explanation as to why infectious diseases claim such a high toll in tropical moist heat. It should prove useful in future studies directed toward the better adaptation of man to his existence problems.

THE DOCTOR IN COURT*

PHILIP J. MCCOOK

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You have listened to a physician and a lawyer on the subject of "The Doctor in Court." As the Judge honored by the invitation to represent the court in this discussion, I am bound to see the problem of each; indeed, if unable to do so, I cannot understand the problem of the litigant and the public either.

I have assumed the Association desires us to present, collectively, each from his own viewpoint, a picture of cases, mainly of trauma, which call for medical testimony. The general title of the symposium is "Some Medico-Legal Aspects of Personal Jury Actions and Actions for Total and Permanent Disability Insurance." But I have been told that it will be satisfactory if I confine my own remarks tonight to the personal injury actions.

In my court, the form in which these are passed upon is mainly that of cases to recover damages. The Trial Clerk of New York County tells me that, as of June 30, 1940, which I consider a fair and representative date, being just before the summer recess, the trial calendar of the Supreme Court carried 5,257 cases, of which approximately 3,904 or about 76⅓ per cent, were so-called negligence suits. Allowing a little for shrinkage from various causes, it may be assumed that 75 per cent of our Law (as contrasted with Equity) cases relate to personal injuries and are likely to call for medical testimony. Further inquiry leads me to believe that one would not find the proportion very different in the Supreme Court for the other four counties of this City, or in our other civil tribunals, that is, the City and Municipal Courts.

The trial lawyer, naturally and properly, sees the function of the doctor one way, and the medical witness sees it in another. The judge, physically and officially overlooking both, and occupying a detached public position, has an excellent chance to study the viewpoint of each, which he must grasp if he is to understand why they react as they do,

* Address delivered February 25, 1941 in the symposium conducted by the Association of the Bar of the City of New York in connection with The New York Academy of Medicine and the Medical Society of the County of New York.

and whether he agrees with either or disagrees with both.

It would be strange, indeed, were the doctor and the lawyer to see eye to eye. One is a scientist; the other, an agent. The former is criticized if he permits himself to become an advocate; the latter, if he permits himself to forget he is an advocate. Yet the doctor, once he has taken the stand on behalf of a party, is scarcely human if he feels no interest in the outcome. This is true even of a subpoenaed attending physician–how much more, of the retained expert. This doctor, employed by one side only, is perhaps in graver and more subtle danger of moral lapse than his legal brother. The barrister, of course, knows that winning for his employer is the best road to professional success, and professional ethics permit and encourage him to follow it. How far the legal profession has at times gone in this direction Lord Brougham is quoted as stating:

"An advocate–by the sacred duty of his connection with his client–knows in the discharge of that office but one person in the world–that client–and none other. To serve that client by all *expedient* means; to protect that client *at all hazards and costs to all others* (even the party already injured), and amongst others to himself, is the highest and most unquestioned of duties."

The author whom I cite for this statement agrees, to be sure, that the great Brougham went too far, saying:

"The sum of the whole matter is, that there is no code of morality applicable especially to any profession or avocation (Harris, Hints on Advocacy [1881], p. 157)."

The medical witness, in addition to the natural bias springing from association with one side only, is not without selfish motive if retained as an expert. He knows he would never have been called were not his views considered favorable to his employer. Yet he is not permitted, by the ethics of his profession, to seek that employer's success. Putting it differently, the doctor, being a witness, is sworn to tell the truth, and when properly questioned must tell the whole truth. The lawyer on the other hand, being an agent only, but also a responsible officer of the court, is not in normal circumstances required to bring out the whole truth by his questions.

Summing the whole matter up again, the lawyer is always an advocate, while the doctor is false to his oath if he becomes an advocate.

From this anomaly stem many misunderstandings and recriminations.

The lawyer is jocularly regarded as a man hired to defeat the ends of justice by delay and the suppression of evidence, the expert sneered at as one who sells evidence and covers his hypocrisy with large language. Yet, I am sure, a great majority of our doctors and lawyers alike desire, above all, to serve justice and truth, and would never dream of deliberate suppression of an important fact.

The explanation is that our system is one of necessary antagonism between conflicting interests, where each side is represented by an advocate required to call fact witnesses, and sometimes other witnesses, supposedly favorable to his side. How the need for expert testimony originally arose and was met has been often and adequately considered, and I shall not attempt to compete with historical articles like that of Learned Hand, written in 1901 (Historical and Practical Considerations Requiring Expert Testimony, Harvard Law Review, Vol. 15, No. 1, p. 40), already discussed by Doctor Heyd.

Judge Hand, treating the subject without special regard to medical evidence, proposed at that time a board of experts, or a single expert, to advise on the general propositions applicable to the case, though he refrained from working out the constitution of such a tribunal. He did not propose to prevent either side from calling experts of their own, with every chance for hypothetical questions and cross-examination. "Only," (he says) "the difference will be that the final statement of what was true would be from the assisting tribunal." He evidently believed that when the assisting tribunal had spoken, there would be, and could be, little doubt where the truth lay.

In 1935, Doctors Elliott and Spillman, of New York City, suggested, in "Medical Testimony in Personal Injury Cases" (2 Law and Contemporary Problems, p. 466), a mixed tribunal of legal, medical and lay judges, rather than the jury of twelve, of whom the physician would have the function of deciding questions as to the "causal relationship between the defendant's act and the injury for which plaintiff seeks damages and as to the extent and permanency of the injuries thus caused." That was quoted in support of a plan said to be essentially similar, by former Chief Judge Crane of our Court of Appeals, in a 1932 address before the Association of the Bar, published in the New York Law Journal of January 29 of that year. All that I can find, however, which Judge Crane said on the subject appears at the end of subsection "V," to the following effect:

"What a speedy disposition there would then be of all these automobile accident cases when the court could appoint arbiters without limit —a lawyer, a doctor, a layman—who would dispose of the cases satisfactorily, yes, more satisfactorily than most of the courts and juries."

The authors, as the title of their paper indicates, are thinking of criticisms of medical testimony in personal injury cases.

In 1936 a report to the California Bar Association, Volume 10, California State Bar Journal, at page 183, after considering the various suggestions for improvement in the practice with relation to the subject, including panels of experts, observed that the latter might be subject to political influence in their organizations and so exclude the best qualified, who, though reluctant generally to testify, "if called on by the court in an individual case, would readily respond." The same year Justice Steinbrink (6 Brooklyn Law Review, p. 155) reached the conclusion (which I understand is the one likely again to be proposed by the commission of which Mr. Kernan is the head) that it is advisable to have a law passed giving the court power to appoint an expert of his own choosing—"a disinterested, competent and licensed physician"—and to tax his compensation as costs (Report of the State of New York Law Revision Commission for 1936). In view of Mr. Kernan's presence here, and what I understand is his intention to discuss the subject, I add only that whatever experience I have had as a judge leads me to the conclusion that it would be unwise, even if possible, to deprive the parties of the right to call their own selected witnesses, including experts.

I have myself on proper occasion, though cautiously, employed the practice of asking the parties in open court to let me name a supplementary or assisting expert. So far, I have never met with refusal, and, as it happens, I have also been able in every case to arrange satisfactorily the matter of my expert's compensation. The experiment has worked well; however, I realize that such uniform good fortune is not likely to continue, and I cannot see any serious objection to having what I am at present able to do only on consent, given the sanction of a statutory power bestowed upon the court.

Dr. Heyd has commented on the practical dangers and difficulties of the expert panel idea. Of course, he knows much more about that subject than I. If such panel, or panels, were to be established by agreement, between responsible medical associations in this city, I am sure that I, for one, would be glad to consider any name on the list, when

indicated, either on consent of the parties, or under statutory powers not now possessed by the justices of my court.

I must venture, however, to differ with him in his doubts whether the judge should be allowed to call his own expert. The only reason he gives against it is, that the doctor most intimate and confidentially close to the judge would probably be his family physician, who may or may not be a medical expert. I confess I would be apt to turn primarily to my family physician for advice where I should go, if I did not already know, to find a general practitioner or specialist suited to my purpose. I submit that if I am to name an expert, as I have been doing on consent, and would like to be able to do as matter of right, I should not be hampered in my choice. Influenced I may be, and must be, by personal considerations, such as the confidence I feel for this, that, and the other physician whom I know personally or by reputation. I suggest that this is no objection, but, on the contrary, a recommendation.

However, before we leave this subject of an expert chosen by the court, I freely state I have found that the conclusions of every doctor I named, like the "assisting tribunal" of Judge Hand, were adopted by the jury; and it is only in case of doubt, where the jury is likely to be confused if an impartial expert is not brought in, that I have ever taken the course of asking consent to such procedure. "If this be treason, make the most of it."

With this confession, that I personally believe no other reform suggested offers great theoretical or practical promise, I pass to the consideration of existing evils, agreeing fully that courts and lawyers cannot permit them to continue unchallenged, but must resolutely seek to eradicate them.

That some of these evils are of long standing, and therefore hard to eradicate, is evidenced by my approval of a list supplied in 1909 by the report to the New York State Bar Association of the Committee on the Regulation of the Introduction of Medical Expert Testimony (32 Report New York State Bar Association, p. 367):

(1) Unsatisfactory standards of experience, inviting charlatanism; (2) partisan, conflicting and hence unreliable character of evidence; (3) loss of time and money; (4) confusing effect on juries of conflicting opinions; (5) unscrupulous hiring by the bar of unprincipled, self-styled "experts"; (6) receipt by trial judges of testimony of incompetent so-called "medical experts." Judge Steinbrink, supra, adds (7) contingent

fees to experts; (8) unequal consequences to rich and poor litigants; (9) the hypothetical question requirement.

Permit me to take them up, one by one, in the light of my own experience.

Expertness is a relative matter. The possibilities of getting the most expert are necessarily limited by considerations such as the financial means of the litigant and the willingness of the best men to testify. Under our New York law, the trial court has full discretion to determine the qualifications of an expert (Slokovitch v. Orient Mutual Insurance Company, 108 N. Y., 56. See also 2 Wigmore on Evidence, section 561). Until he finds a witness qualified, he has the power to exclude his opinions altogether. A judge naturally hesitates, however, to deprive a litigant of evidence which the latter needs, slight though its value may be: for example, that of a young and inexperienced attending physician, who nevertheless, by reason of required modern training and intelligence is able to furnish some kind of an opinion. The jury is always told in that event, and probably should be told in every case, that such testimony is only advisory. Indeed, as you know, the jury may reject any expert's testimony if they like (though not capriciously) which constitutes a further and final protection.

The partisan character of expert testimony is inseparable from our present system, as I have already shown, and I do not regard it as a fatal objection. If too obvious in the case of a particular witness, partisanship defeats itself. If present in one side, it will probably also appear on the other, and the jury is quick to size up the situation. I certainly agree with the famous saying in 1856 of Lord Chief Justice Campbell, in Rex v. Palmer, quoted in Hist. Crim. Law of England (1883) Vol. III p. 389:

"It is, in my opinion, indispensable to the administration of justice, that a witness should not be turned into an advocate, nor an advocate into a witness."

He meant, no doubt, that wise and honest barristers and physicians, with the help of the court and the jury, could and would coöperate to that end. Such also is my hope—and more than a pious one.

No doubt expert testimony does prolong trials, but good evidence is worth taking time for, and what we are trying to work out is the problem of getting good evidence. In the old days, hypothetical questions were terrible time-wasters. My observation is that this particular

evil is already passing. The rules of form are not so strictly observed by lawyers or required by judges, and courts are quicker than formerly to interfere and cut through to the heart of the matter with questions or suggestions of their own, before the situation becomes acute. I may admit frankly that hypothetical questions bothered me greatly in my earlier days on the Bench; but many years have passed since such a question has caused serious difficulty in any case which I have tried. No doubt my associates would say the same thing.

It is also true that juries are occasionally confused by expert testimony, contradictory in character. I have already explained how I deal with such conditions myself. As a rule, the jury will see through the fog, separate the wheat from the chaff, and either reject the whole thing (as they have the right to do), or pick and choose, which is equally their right. I am often comforted by the reflection—which seems to have occurred to Doctor Heyd also—that perhaps medical expert testimony as contrasted with medical fact testimony is not really quite as important as the experts themselves believe.

It is true that an unscrupulous doctor will always find an unscrupulous lawyer to hire him. Every profession has its parasites. Delousing is a tedious and unpleasant process, which neither your profession nor mine can escape.

I do not like to let pass unchallenged the very serious charge of unequal consequences to the rich and poor. I have already hinted that this is a consideration, how important I do not venture to say. From the same source already quoted, I have it that in 67 per cent of the personal injuries cases on the June, 1940, calendar of my court, the defendant is represented by an insurance company. It is common knowledge that in the majority of all cases the defendant has greater resources than the plaintiff. This is always present (perhaps more often than proper) in the minds of court and jury alike. The judge has many an opportunity to meet the difficulty, and if the law favored by Mr. Kernan's Committee is passed, he will be able to act oftener. I do not know whence the opposition to that law in the past came, but whether it was from the plaintiffs' or the defendants' side, I venture to suggest that a policy which would continue to oppose is short-sighted. Inequality is an unescapable fact, and not confined to cases where expert witnesses are used; it runs right through every system of law, and, indeed, every other system. The wise and the just must be watchful and must be astute,

at every turn, to minimize it.

I think I have mentioned, directly or indirectly, all the abuses on this particular list, except contingent fees. An expert witness is often asked by opposing counsel whether his compensation depends on the result. I have no way of knowing how often that in fact is the basis of the expert's retainer. The matter seems primarily the affair of the medical profession. We lawyers have troubles of our own along that line.

So far I have spoken mainly of the expert medical witness, because the discussion seemed to take that turn. The attending physician is the man who plays by far the more important role. To the ordinary litigant in a personal injury trial, opinion evidence, as I have pointed out, may be a luxury; fact evidence is a necessity. Consider a laborer who has broken his leg in an accident and lost two months' wages. No matter how rank the negligence which caused his injury, he will not win unless the doctor who attended him appears in court to testify. On the other hand, maybe the injury has been feigned and a hospital intern knows that fact. If he is not brought to the stand, imposition on an innocent defendant results. Probably the majority of subpoenas in New York County, served on doctors, go to men like that, men whose presence is absolutely essential to a just outcome. Now, since a case in the Supreme Court of this County takes one and a half years to reach, what happens? Almost invariably the doctor has long since forgotten all about the case. To respond means loss of time and money, yet respond he must. He is nearly always exasperated, and sometimes defiant. Nobody can blame him for that.

I can remember when there seemed to be a standing feud between certain trial lawyers and the practicing physicians of this town, over such a situation—one group inconsiderately subpoenaing right and left, naming without alternative a certain day and hour, the other evading or even ignoring service. I am sure matters are better now. At all events, there is not a judge on my bench who would not, if properly approached, require trial counsel to show a medical witness every consideration. If, for example, the latter promises to come when called, he will not be disturbed until actually needed. More than that, the judge will always, when possible, permit his testimony to be taken out of turn, as a matter of additional convenience.

I hesitate to tell any practitioner his duties, but perhaps I may be permitted to quote what Dr. J. W. Courtney said to a graduating class

of medical students, in 1915 (Medical Testimony in Personal Injury Cases, supra):

"The present mode of procedure in our courts, in so far as medical testimony is concerned, is not a particularly edifying one. To illustrate this point, let us take, for example, a case of the type which is most commonly met with in everyday work of the courts—an action of tort for personal injuries. In such a case the plaintiff is practically always of the proletariat class; the defendant, a public service corporation or an insurance company. The army of witnesses on either side is generally appalling. Of these the medical ones alone concern us. They are of two hostile camps, and prepared to attempt, under solemn oath, to uphold opinions diametrically opposed, yet supposedly derived from a single series of facts and observations.

"The situation is a deplorable one, and nobody discerns the glaring wrong of it all with clearer vision than certain high-minded men from our ranks, who have long striven to procure legislative enactment looking toward the abolition of this evil.

"To me, for many reasons which I cannot here enumerate, it seems hopeless to expect that legislative appeal on the part of such men will ever be fruitful of the desired results. Hence it is the bounden duty of every man in the profession so to shape his conduct toward cases which promise to eventuate into court proceedings, that due respect will be given his opinion, that he will not merit the biting sarcasm, the sneers, the raillery and general brow-beating of opposing counsel. And, most of all, that through his efforts the ends of justice will really be accomplished."

Dr. Courtney is dead, but his words, spoken so long ago, still live and merit our careful attention—doctors, lawyers and judges alike. While he was speaking principally of experts, what he said applies in large part to the medical fact witness as well, and is broad enough to include the duty of the latter while he is attending a patient who has suffered an accident and therefore is likely to call him to court some day.

To illustrate from one of my own cases: A plaintiff blamed serious injuries on the defendant's carelessness in drawing off the water from a public pool in which he had dived. His medical evidence aroused my suspicion. The doctor, who certified he had attended the plaintiff, was very vague as to the exact date, and he had no original records with

him—merely notes from such records. I adjourned the trial to await his return the next day. He appeared with records which showed concluisvely that this patient had been treated by him long before the date of the alleged accident.

I hope the doctors present grasp the seriousness of such an incident. Of course, the jury found for the defendant in a few minutes. Then I had the unpleasant duty of holding a miserable, irresponsible and, as he turned out to be, an habitually drunken plaintiff for the grand jury, which indicted him for his flagrant perjury, and he was sent to prison. The doctor escaped any punishment except whatever humiliation he endured in court, and, I regret to say, he may have been too thick-skinned to feel much of that. Yet it is plain he, like plaintiff's lawyer, had neglected his duty. Each thereby lowered, in open court, the dignity and standing of his own profession.

They had done one service to me, in uncovering what appeared to be a general fault of private practitioners of medicine—neglect in keeping adequate original records, and failure as matter of routine, perhaps because of the scanty nature of their records, to bring whatever they have to court. I am well aware what excuses may be offered, but from the judge's standpoint they are inadequate, and reflection on your part will show we are right.

Next to the complaint that the lawyers and judges waste their time, I hear most frequently that we permit doctors to be imposed upon by compelling them, through the receipt of no more than a subpoena fee, to testify as expert witnesses. Indeed, Dr. Heyd has told you that they are subject to "chiseling" by the plaintiff's lawyer, which indicates his belief that this is a general practice. He states that in New York a physician may not be called upon to give opinions, or to exercise the function of an expert witness, without compensation or agreement for compensation. My own practice has been to regard any such attempt as an abuse of the subpoena, and when appealed to I have generally sustained the witness in his refusal to act as an expert unless reassured. Sometimes, indeed, I have gone so far as to interfere without such appeal (People v. Raizin, 211 App. Div. [2d], 446 at 461-2).

I have tried to examine the criticisms in which we are particularly interested, and discuss them frankly. The medical profession has a just grievance wherever its members are treated discourteously or unfairly in court, which is indefensible. I hope I have explained some of your

difficulties and allayed some feelings of suspicion.

Your Committee, gentlemen of the Bar Association, have performed an act professionally graceful and socially useful in inviting us to meet in this symposium to discuss problems of common interest. To you, representatives of the medical profession and your several learned societies, I owe thanks for the opportunity of exchanging ideas on a subject which affects the courts so deeply.

Yearly, monthly, weekly, wise words issue from the rostrum of the Medical Association and the Bar Association all over the United States. The citizen sighs with impatience as he reads about these proceedings in the next day's paper, for the same sheet brings news of dirty work at the cross-roads which the speeches of the pundits may have let pass, perhaps with perfunctory condemnation. We cannot afford to ignore this popular criticism, to which honest lawyers and doctors, anxious of course to make a living, but even more anxious to obtain and deserve public commendation, are extremely sensitive. Imagine, if you can, the influence on legislation and public opinion of such a multitude, if it could be exercised in unity, armed with the minimum of self-interest and the maximum of interest in the public welfare.

We both tend to pontificate, lawyer and doctor alike. It is in our blood. When the priest announced "Thus saith the Lord," he proclaimed laws of Divine origin, physical, social and moral, with little distinction, at first, among them. Thus were born the necessity and opportunity for the lawyer and doctor, with their priestly origin and their priestly responsibilities. You think, I know, that through our supposed power with the three branches of government, we lawyers have the final say, even in matters within your province. Alas, neither of us seems to have much power left nowadays, and it may be that ere long none will be left unless we have all, by that time, become public officials ourselves. So let us continue, while we may, to take counsel together, as equals.

In fact, you have the edge. You are the doctor, the learned one, which is the meaning of your title. When institutions of learning wish to honor a lawyer, they call him "Doctor of Laws." You are, through man's infirmities, the master of masters, and you have declared yourself, in your Hippocratic oath, the servant of servants. It is you who rule at the death bed, where we all arrive at the last—you and the priest. We turn to you because your knowledge makes you stronger than we, and

because your principles compel you to attend us even when the lawyer no longer finds anything to interest him. Continue to be merciful.

You have your oath; we have canons of ethics, of which William Travers Jerome once said, in my hearing, that there never was a problem of legal ethics which a child could not settle in a moment once you take the dollars and cents out of it. There is more than a little truth in that. Maybe we must read your regulations also in the light of human frailties.

Doctors, and not lawyers, are the final judges of medical ethics. Each of you carries about, within him, his own best informed and severest critic. Do not lean too heavily on codes and canons. They may afford satisfaction to the mercenary individual seeking an alibi and looking for loopholes. They supply cold comfort to the conscientious.

Gibran, poet and prophet, said:

"He who wears his morality but as his best garment, were better naked.

The wind and the snow will tear no holes in his skin.

And he who defines his conduct by ethics imprisons his song-bird in a cage.

The freest song comes not through bars and wires."

The important thing is that somebody should set an example to the rest of us in maintenance of ideals, kindness to the afflicted, enlightenment of viewpoint, avoidance of prejudice, and most of all, courage in action. I do not seek to minimize the disappointment, weariness and self-sacrifice entailed. But having carried the torch through the Dark Ages and being, as your representative has admitted to-day, in a better economic position to meet emergencies than we lawyers, you are not likely to falter now that you have in so large a measure the public ear and the confidence of the courts.

THE FOUNDING OF THE NEW YORK LARYNGOLOGICAL SOCIETY

SOLOMON R. KAGAN

AMERICAN medical men have been pioneers both in scientific and organizing medicine. As Welch said, "American medicine became independent and took a lead in various aspects of medical sciences." A significant achievement of New York pioneer work may be considered the foundation of the New York Laryngological Society on October 13, 1873. Clinton Wagner of New York City, a foremost surgeon, who studied laryngology two years abroad, foresaw a great future for this specialty. In 1873 he instituted the Metropolitan Throat Hospital in New York City. This was the first institution in the United States which treated exclusively diseases of the nose and throat. In October of the same year he brought together a group of physicians who held positions in throat clinics in New York City for the purpose of organizing a society "not only for mutual improvement and the advancement and enlargement of their limited knowledge of the subject, but for the purpose of establishing on a solid footing with the medical profession at large the specialties of rhinology and laryngology in this country." At this meeting the New York Laryngological Society was established by the following doctors: Clinton Wagner, Frank H. Bosworth, Geo. M. Lefferts, Morris J. Asch, Woolsey Johnson, Horatio Bridge, Charles McBurney, Robert F. Weir, Matthew D. Mann and Francis P. Kinnicutt. Robert F. Weir was elected president; Clinton Wagner, vice-president; and Woolsey Johnson, secretary. Other original resident members of this Society were: Andrew H. Smith, Frederick A. Burrall, Beverly Robinson, and Samuel B. St. John, Jr.

Among these organizers were well-known surgeons who contributed important work to their specialty. Thus, M. J. Asch invented the "Asch operation" for a deviated septum; F. H. Bosworth, author of a classical book on laryngology (1881), placed laryngology on a firm scientific basis; G. M. Lefferts, in collaboration with J. Solis-Cohen, L. Elsberg and Knight, founded the *Archives of Laryngology* in New York in

1880. The activities of the Society were manifold. It stimulated research work in a new field of medicine. Rare cases of interest were presented at its meetings for examination and discussion. Discoveries of new theories and treatments, inventions of useful instruments and new methods and ideas pertaining to laryngology were reported and widely discussed and clarified. Dr. Bosworth had read sections of his work, *Nose and Throat,* before the members of the Society who expressed their criticism and opinions. Sir Morell Mackenzie of London read before this Society his paper "Hemorrhage after Tonsillotomy."

The New York Laryngological Society was the first society of laryngologists in the world, and served as a stimulus to the profession for the advancement of medical specialism. Five years later, Frank Davis of Chicago proposed the foundation of a National Society of Laryngologists. It was founded on June 3, 1878, in Buffalo, New York, known as the American Laryngological Association, which also was the first national association of laryngology in the world. Dr. Wagner's idea spread abroad, and fifteen years after the foundation of the New York Laryngological Society, Sir Morell Mackenzie founded in London the British Laryngological Association (1888). Dr. Mackenzie was the first man in England to put specialization on a sound scientific footing and founded the first special hospital, the Golden Square Throat, Nose and Ear, in London.

The difficulty of the foundation of the Society was greatly due to the fact that many physicians of that time looked askance at all specialties. In England at that time specialism was rather frowned upon by the orthodox leaders of the profession. In America this antagonism was markedly expressed by many practitioners who barred the specialists as not being honest and reliable practitioners. In connection with this, it is of interest to tell the fact that some members of the Academy of Natural Sciences of Philadelphia declared that the eminent laryngologist Jacob Solis-Cohen of Philadelphia was engaged in unethical practice. When Solis-Cohen was in the amphitheatre during a surgical clinic at the Jefferson Medical College, the distinguished surgeon, Samuel D. Gross, introduced him to the class as a man fast leaving the ranks of legitimate practice to become engaged in a narrow specialty. "Why," Gross said, "he devotes most of his time to a cubic inch of the human anatomy. Some day, I suppose, we will have specialists confining themselves to diseases of the navel."

Wagner's pioneer work was therefore of particular value, as he was among the forerunners of the development of specialism in this country. For over ten years the Society was in existence and accomplished much. As the older members, the organizers of the Society, died, the progress of the Society ceased. The last important meeting took place at the home of the last President of the Society, Rufus P. Lincoln, in 1883. He failed to call the usual annual meeting for the election of officers, and while retaining his position as president he died. In 1885, at the suggestion of Abraham Jacobi, the Society was merged with the Laryngological Section of The New York Academy of Medicine.

The Book of Minutes of the Society which contained a complete history of all the work of the New York Laryngological Society was lost at the home of the last President of the Society. I have in possession three letters written by the secretary and two organizers of the Society. These letters throw some light on this pioneer Society and contain a list of its original members.

On February 7, 1874, Morris J. Asch wrote to Jacob Solis-Cohen of Philadelphia:

"You know perhaps from Dr. Robinson that we have formed a Laryngological Society—of which I will write you more fully in a few days. Today I want to know from you if it would be agreeable to you to be elected a corresponding member and let me know so that I can have you elected at the monthly meeting on Thursday next. Otherwise I may have to wait a couple of months perhaps. Anyhow, if I do not hear from you I will propose you. Where can I find the best authority on Naso Pharyngeal Catarrh?

Many thanks for your kindness to Robinson. The throat hospital is in full blast. In a few days I will write you all about it. Today I am in great haste—& stopped in here so as to be sure to have my letter go."

In the same year the secretary of the Society, Woolsey Johnson, sent the following note to Solis-Cohen:

"230 West 43d—St.
New York, Mch 15, 74.

J. Sollis Cohen, M.D.

Dear Doctor:

I have the honor to inform you that at a regular meeting of the New York Laryngological Society, held on the 12th inst., you were elected an honorary member.

The Society is an association of professed Laryngoscopists resident in this city, and meets on the 2d Thursday of every month for the purpose of "promoting the study of affections of the larynx, pharynx and adjacent parts."

The number of honorary members is by the constitution of the Soc. limited to ten.

Hoping that you will accept the membership thus offered, and that you will be able frequently to be present at our meetings, I am, Doctor, with assurances of my own high regard,

Very respectfully, your obt svt
Woolsey Johnson, Secretary"

Four years later, Dr. Bosworth, one of the organizers of the Society, wrote to Solis-Cohen:

"26 West Forty-Sixth Street
Friday, Oct. 4th–'78

Dear Doctor:

I have just learned that the next meeting of the N. Y. Laryngological Soc. is called at my house on Oct. 10th, and also that there is a prospect of your being present–I need not tell you what a genuine pleasure it will be to all of us to meet you at one of our gatherings, and I trust that nothing may interfere with your coming.

It occurred to me to write to you and ask that you will suggest to me anyone that you would care to meet, and I will only be too glad to extend to him an invitation to be present at the meeting.

Our society has been fairly prosperous since its organization and we have done some good things I hope. Still I should look for a new interest and enthusiasm to be infused into it, by the presence of one who has done so much in the country for Laryngoscopy–and should like to make your coming the occasion of awakening a something better of individual interest on the part of our members.

The meeting is given up generally to the consideration of clinical cases presented, rather than to the reading of formal papers and for next week there is no special work laid out. I need not say that we should be glad to listen to anything that you might bring with you–

With regards
Very truly
F. H. Bosworth"

Short was the life of the New York Laryngological Society, but its accomplishments were significant. It opened a new path in medicine, creating an organization for the advancement of laryngology. It contributed to the introduction of novel ideas, clarification of problems pertaining to this specialty and popularization of new laryngological methods and inventions discovered here and abroad. Many scientific papers were read and thoroughly discussed by able men at the meetings of the Society. The latter served as a valuable ring in the chain of organized medicine.

New York City was the birthplace of laryngology. Horace Green of New York, the father of laryngology, was the founder of laryngology in the United States, and the New York Laryngological Society was the first laryngological society in any part of the world.

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"Possession does not imply approval"

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NOTICE

IDENTIFICATION OF INDIVIDUALS IN GROUP PHOTOGRAPHS

The Librarian invites the assistance of Fellows of the Academy in establishing the identity of individuals in a number of old photographs of groups of medical men.

PROCEEDINGS OF ACADEMY MEETINGS

STATED MEETINGS

OCTOBER 2—*The New York Academy of Medicine.* Executive Session—a] Reading of the Minutes; b] Introduction of Amendment to By-Laws. ¶ Papers of the evening—a] Memorial to Dr. Frederick Banting, Charles H. Best, Director, Department of Physiology, and Banting and Best Department of Medical Research, University of Toronto; b] The use of insulin in its various forms in the treatment of diabetes, Elliott P. Joslin, Medical Director, George F. Baker Clinic, New England Deaconess Hospital, Boston; c] Prevention of diabetes from the experimental viewpoint, Charles H. Best

OCTOBER 30—*The Harvey Society (in affiliation with The New York Academy of Medicine).* The First Harvey Lecture, "Nervous Activity and Visual Mechanisms," H. Keffer Hartline, Assistant Professor of Biophysics, Johnson Research Foundation, University of Pennsylvania.

SECTION MEETINGS

OCTOBER 3—*Surgery.* Reading of the minutes. ¶ Presentation of cases—a] Cases illustrating first paper of the evening, Henry H. Kessler (by invitation); b] Cases illustrating second paper of the evening, A. G. Fuller (by invitation). ¶ Papers of the evening—a] Amputations of the upper extremity (motion picture), Henry H. Kessler (by invitation); b] Amputations of the lower extremity, A. G. Fuller (by invitation); c] Use of local refrigeration in extremity surgery, Lyman Weeks Crossman (by invitation), Frederick M. Allen. ¶ General discussion. ¶ Executive session.

OCTOBER 7—*Dermatology and Syphilology.* ¶ Presentation of cases—miscellaneous. ¶ General discussion. ¶ Executive session.

OCTOBER 7—*Combined Meetings, Section of Neurology and Psychiatry and the New York Neurological Society.* Reading of the minutes. ¶ Papers of the evening—a] Presidential Address, A. A. Brill; b] The electrofit in the treatment of mental disease, David J. Impastato (by invitation), Renato Almansi (by invitation); Discussion by S. Bernard Wortis; c] Psychiatric disorders in forty men teachers, Edward B. Allen (by invitation); Discussion by Edwin J. Doty (by invitation), Emil Altman.

Pediatrics—Because of the annual meeting of the American Academy of Pediatrics in Boston the October meeting of the Section was not held.

OCTOBER 28—*Obstetrics and Gynecology.* Executive Session—a] Reading of the minutes; b] Program presented by the Obstetrical Department of the Bronx Hospital. ¶ Presentation of cases—a] Gestation neuronitis, A. C. Posner (by invitation); Discussion by Harry Aranow; b] Pregnancy after prolonged sterility; change of life, M. Rosensohn; c] Central placenta praevia—hyperthyroidism—vaginal delivery—thyroid crisis, Israel Kibel (by invitation); Discussion by H. C. Williamson. ¶ Papers of the evening—a] Further studies in soft tissue diagnosis in pregnancy by x-ray, W. Snow; Discussion by Paul Swenson, Presbyterian Hosp.; b] An outbreak of impetigo. Studies by the hospital epidemiologist, W. Wolarsky (by invitation); Discussion by J. Felson. ¶ General discussion.

Genito-Urinary Surgery, Medicine, Ophthalmology, Otolaryngology, Orthopedic Surgery—These Sections held no meeting in October because of conflict in dates with the Graduate Fortnight.

IN MEMORIAM

Ludwig Kast

With profound sorrow The Bulletin records the death of Ludwig Kast, honored Fellow of this Academy and devoted friend of medical education, who in the year 1927 proposed to the Committee on Medical Education of the Academy the institution of an annual graduate fortnight dedicated to the complete exposition of a chosen medical subject for the benefit of practitioners of medicine.

We can conceive of no more fitting tribute to Doctor Kast than the publication of the following letter from his own hand:

[Letter from Dr. Ludwig Kast proposing inauguration of Graduate Fortnights to be undertaken by the New York Academy of Medicine]

February 28, 1927

Doctor James F. McKernon,
Chairman, Committee on Medical Education,
New York Academy of Medicine.
Dear Doctor McKernon:

In the course of an investigation concerning the future development of Graduate Medical Education in New York, it has occurred to me that to the progressive efforts which the Academy has lately inaugurated, a new feature could be added which might be planned as follows:

A month is chosen during which all available opportunities in New York for lectures, demonstrations, symposiums, courses, ward rounds, operations are coordinated for the purpose of covering some big, outstanding problem of clinical nature from as many angles as possible. The underlying purpose would be to feature the topic by the very mass of contribution and by its quality so as to make it an event of national and later of international importance. After this has been accomplished a few times it will, as a matter of course, establish that month as a standing feature of Medical Progress in New York.

1. *Time:* October or May would seem the months during which the largest number of physicians could be expected from out of town and also from within a radius of one hundred miles of New York. Between these two months the preference might be for October as those who would actively take part in this plan would be still refreshed from the summer.

   For the first two weeks most of the important lectures and presentations could be planned, to be overlapped by two weeks more of courses and seminars and two or three important meetings toward the end of the month.

2. *Subjects:* Some such problems as:
   The Cancer problem
   Infectious diseases and Problems of Immunity
   Heart and Arteries
   The problems of old age
   The problems of ductless glands in Medicine and Surgery
   Tuberculosis
   Preoperative and postoperative management in abdominal surgery.

3. *Program:* (a) Special meetings at the Academy with symposium on the particularly debatable aspects of the problem.
   (b) Cooperation if possible by the Harvey Society, Herter lectures, etc.
   (c) Meetings of the larger medical Societies in New York with papers pertaining to the chosen subject.
   (d) Graduate courses at the medical colleges, Polyclinic, Post-Graduate, etc. Also Seminars on the subject.
   (e) Outstanding authorities from Europe and from the Americas could be invited to read papers, particularly those who are already secured or to be secured for the Herter lectures, Harvey Society, etc.
   (f) National societies, having for their object the advancement of the chosen subject might be induced to hold their annual meeting in New York during that month.

4. *Preparations:* It will, undoubtedly, take not less than half a year to make proper affiliations and coordinate the available material and to publish the program, at least its outstanding papers and courses, well in advance.

   In arranging the program it might be well to have the graduate courses handle the simple, practical or didactic parts of the program and to reserve for the large meetings those parts which would lead to real debate particularly on the borderlines of the subject and its bearing upon Public Hygiene, and in general upon the needed cooperation between the medical profession and our Commonwealth. For this purpose the Academy might invite speakers from outside the medical profession.

5. *Publication:* It would seem possible to secure a nation-wide publicity well in advance without any expense.

   The Academy, with or without cooperation of a publishing house, might undertake to publish a volume covering the important parts of the transactions.

Sincerely yours,

L. KAST

DEATHS OF FELLOWS

BISHOP, LOUIS FAUGERES, SR.: 121 East 60 Street, New York City; born in New Brunswick, New Jersey, March 14, 1864; died in New York City, October 6, 1941; received his B.A. degree from Rutgers University in 1885 and graduated in medicine from the College of Physicians and Surgeons, Columbia University, in 1889; elected a Fellow of the Academy March 2, 1893; and served the Academy as Secretary from 1897 to 1903.

Dr. Bishop was consultant physician to the Lincoln Hospital, the Sea View Hospital and Goshen Hospital at Goshen, New York; director for the New York School for the Deaf; and instructor in graduate courses in internal medicine at Columbia University and Bellevue Hospital. At one time he was professor of heart and circulatory diseases at Fordham University School of Medicine and consulting physician to the Mercy Hospital at Hempstead. He was a diplomate of the American Board of Internal Medicine, a Fellow of the American College of Physicians, a Fellow of the American Medical Association, past president of the American Therapeutic Society, a member of the National Gastroenterological Association, a member of the American Association of Pathologists and Bacteriologists and a member of the County and State Medical Societies.

Dr. Bishop was the author of "Heart Disease, Blood Pressure and the Nauheim Treatment," "Arteriosclerosis," "Heart Troubles, Their Prevention and Relief," "A Key to the Electrocardiograms," "A History of Cardiology," "Mechanism of the Heart and Its Anomalies," (translated from the French), and numerous articles.

CONNELL, KARL: Wintoon Lodge Branch, New York; born in Omaha, Nebraska, July 4, 1878; died in Scarsdale, New York, October 18, 1941; graduated in medicine from the College of Physicians and Surgeons in 1900; elected a Fellow of the Academy, October 6, 1904.

Dr. Connell was professor of surgery at Creighton University, Omaha, from 1919 to 1924. As a Major in the A. E. F. in France, Dr. Connell won the Distinguished Service Medal by designing the first American gas mask. He served as U.S. Government observer in Germany and Austria in 1915. After the war he founded the Presbyterian Hospital at Omaha and was its president until 1924. He was a Fellow of the American College of Surgeons and a Fellow of the American Medical Association.

GREGORY, MENAS SARKAS: 115 East 61 Street, New York City; born in Morach, Turkey, July 14, 1872; died in Tuckahoe, New York, November 2, 1941; graduated in medicine from the Albany Medical College in 1898; elected a Fellow of the Academy December 3, 1908.

Dr. Gregory was director of the psychiatric department of Bellevue Hospital from 1904 to 1934 and consultant in psychiatry to that institution from 1936 until his death. He was consultant in psychiatry to the New York Neurological Institute, a former professor of psychiatry at the New York Post-Graduate Medical School, and professor emeritus to the New York University College of Medicine. He was a diplomate of the American Board of Psychiatry and Neurology, a Fellow of the American Medical Association, and a member of the American Psychiatric Association, the American Neurological Association, the Association for Research in Nervous and Mental Diseases, and the State and County Medical Societies.

REGAUD, CLAUDE: Paris, France: born at Lyons, France, January 31, 1870; died at Couzon-au-Mont-d'Or, France, December 28, 1940; elected an Honorary Fellow of the Academy, November 8, 1934.

Dr. Regaud was professor at the Institut Pasteur; director of Laboratoire de Radiophysiologie de l'Institut du Radium and of Service Thérapeutique de La Fondation Curie at Paris; a member of the Institut de France, a member of the Académie de

Médecine de Paris and an honorary member of the Académie Royale de Médecine de Belgique. During the World War he joined the Army with the rank of Captain in the Medical Corps and was promoted to the rank of *Médecin Commandant*. He was decorated with the Cross of the Légion d'Honneur and the Croix de Guerre.

Regaud and his school exerted an enormous influence on cancer research. Foreign governments and numerous institutions of both hemispheres turned to him for counsel. In 1924 he delivered an address at the 9th Annual Meeting of the American Radium Society in Chicago on "Some biological aspects of the radiation therapy of cancer," which was published in the American Journal of Roentgenology. In 1928 he was a delegate to the International Congress of Radiology, and in 1934 he was chosen to deliver the memorial address for Mme. Curie, at the Fondation Curie.

Dr. Regaud was the Editor of *Leçons de chirurgie de guerre* and the author of numerous studies, among which are "Traitement du cancer du col de l'utérus par les radiations" (1926); and "Comment on peut concevoir actuellement l'organisation de la lutte contre le cancer" (1929). Together with his collaborators, he published numerous works of which the *Archives de l'Institut du Radium*, a series of studies devoted to radiophysiology and radiotherapy, is regarded as a masterpiece.

SELIG, SETH: 17 East 96 Street, New York City; born in New York City, October 31, 1897; died in New York City, November 2, 1941; graduated in medicine from the College of Physicians and Surgeons, Columbia University, in 1921; elected a Fellow of the Academy, February 4, 1932.

Dr. Selig was attending orthopedic surgeon to the Montefiore and Mt. Sinai Hospitals, and a medical officer of the New York Fire Department. He was a diplomate of the American Board of Orthopedic Surgery, a Fellow of the American Medical Association, a Fellow of the American College of Surgeons, a member of the American Academy of Orthopaedic Surgeons and a member of the State and County Medical Societies.

SHINE, FRANCIS WAYLES: 116 East 62 Street, New York City; born in Orlando, Florida, June 25, 1875; died in Charlottesville, Virginia, September 24, 1941; graduated in medicine from the University of Virginia in 1898; elected a Fellow of the Academy May 5, 1910.

Dr. Shine was consulting surgeon to the New York Eye and Ear Infirmary and a member of its board of directors; consulting ophthalmologist to the New York Hospital; a diplomate of the American Board of Ophthalmology; a Fellow of the American Medical Association; and a member of the American Academy of Ophthalmology and Otolaryngology, the American Ophthalmological Society, and the State and County Medical Societies.

Dr. Shine served in France as a Major with the American Expeditionary Force unit formed by the New York Hospital in 1917-18 and later served as consulting ophthalmologist for our Army in the Paris area.

WHEELWRIGHT, JOSEPH STORER: 654 Madison Avenue, New York City; born in Bangor, Maine, November 23, 1875; died in Long Point, Ontario, Canada, October 9, 1941; received the degree of A.B. from Yale University in 1897; graduated in medicine from Cornell University Medical College in 1900; elected a Fellow of the Academy, January 7, 1926.

Dr. Wheelwright was founder and consulting surgeon to the Southampton Hospital at Long Island, and consulting surgeon to the Presbyterian Hospital in New York City. He was one of the founders of the Doctors Hospital, and, at the time of his death, was one of its directors and secretary of its medical board. At one time he also was connected with the Neurological Institute of New York and the East Maine General Hospital in Bangor. He was a Fellow of the American College of Surgeons, a Fellow of the American Medical Association, and a member of the State and County Medical Societies.

During the World War, Dr. Wheelwright served twice overseas, first as an executive of a French Army hospital unit and later as a Major in the Medical Corps of the American Expeditionary Force.

CITY OF NEW YORK
DEPARTMENT OF HEALTH
BUREAU OF RECORDS

125 WORTH STREET, NEW YORK, N. Y.

October 27, 1941

TO THE PRIVATE PRACTITIONERS OF MEDICINE AND SUPERINTENDENTS OF CIVIL HOSPITALS IN THE CITY OF NEW YORK

Subject: *New Procedure Regarding the Completion of Death Certificates*

Commencing November 1, 1941, private practitioners of medicine and civil hospital authorities will be relieved of the responsibility of completing the left side of death certificates required to be filed with the Department of Health, except in those instances where the body is unclaimed by a funeral director and is delivered to representatives of the City Mortuary for burial in the City Cemetery.

This procedure was adopted by the Board of Health, after approval by (1) the Public Health Relations Committee of the Academy of Medicine, (2) the Coordinating Council of the Five County Medical Societies, (3) the Greater New York Hospital Association, and (4) the Metropolitan Funeral Directors Association.

Funeral directors will hereafter be held responsible (1) for obtaining and entering the personal particulars required to complete items 2 to 14 of the death certificate, and (2) for obtaining the signature and address of the Informant, and relationship to the deceased in item 15. This change will bring procedures in New York City more nearly in conformity with practices in this regard elsewhere in the United States and will relieve physicians and hospital authorities, who frequently have little or no interest in this information, of this responsibility with which they were previously charged.

This means that, with the exception of City Cemetery cases—mentioned above—physicians and hospital authorities will be required only (1) to enter the name and social security account number of the deceased in item 1, (2) to complete the Medical Certificate of Death—items 16 to 21, inclusive, (and in Manhattan the Physician's Confidential Medical Report) and (3) to fill in the Physician's Supplementary Certificate of Death by Natural Causes—on the back of the certificate, or on a separate form, *except* in connection with Form 17-H-1941, used in Manhattan only (see upper left hand corner of certificate.)

The Board of Health is confident that this change of procedure will receive unanimous approval and trusts that private practitioners and hospital authorities will renew their efforts to prepare *promptly, completely,* and *accurately,* the medical certificate of death and the related documents for which they are still responsible.

It would be appreciated if a copy of this letter could be posted on hospital bulletin boards where it will come to the attention of all attending and visiting physicians, as well as staff members.

THOMAS J. DUFFIELD,
Registrar of Records.

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